THE CHICAGO MEDICAL SCHOOL
QUARTERLY

VOLUME 17, NUMBER 2 FEBRUARY, 1956

CONTENTS

ORIGINAL ARTICLES

TRAUMA

I. The Physiological Basis of Burn Therapy 49
Harry H. LeVeen, M.D.

II. Ocular Injuries 59
Paul Sternberg, M.D. and Burton M. Krimmer, M.D.

III. Management of An Acute Head Injury 66
Milton Tinsley, M.D.

IV. Trauma to the Abdomen 71
Ellsworth E. Hasbrouck, M.D.

V. Management of Acute Dislocations of the Extremities 76
Sydney H. Morgenstern, M.D.

SPECIAL ARTICLES

Current Views Regarding Collagen Diseases and Pregnancy 81
S. J. Turner, M.D.

Chemical Tests for Intoxication 87
Daniel T. Dragel

FEATURES

School Notes and News 93

Abstracts 96

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THE PHYSIOLOGICAL BASIS OF BURN THERAPY

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Thermal injury at the body surface is the usual type which is seen in burns. However, the heat may be generated by high frequency sound waves (sonar), absorption of ultra high frequency, electromagnetic waves such as radar and infra-red absorption. In each case the lethal effects are non-specifically caused by heat. The destruction of skin need not be complete and the most commonly used classification relates the depth of burns in three degrees. The first degree burn produces only erythema. The second degree burn produces destruction of the skin and blistering, but the full thickness of the skin is not destroyed. In a third degree burn the full thickness of the skin is destroyed; the skin may have a dead white appearance and is often leathery on palpation. It is not strange that the most painful burns are first and second degree burns, since the nerve endings in the skin are completely destroyed with third degree burns. The depth of the burn will depend upon the intensity and the duration of exposure.

Scalding with hot water usually causes second degree burns, while third degree burns are most common when the damage is caused by fire or flames. Some burns are uniformly shallow and are confined to the exposed portion of the body following an explosion or an intense, sudden liberation of heat. These burns are called flash burns, and are rarely third degree burns. In such cases, the injury is usually caused by absorption of infra-red radiation and the absorption is complete. This type of burn occurs with atomic explosions. The color of the clothing will greatly influence the protection to the intense infra-red radiation since dark clothing reflects less infra-red rays, whereas light clothing has a high degree of reflection. In some cases the infra-red absorption by dark clothing is sufficient to ignite the clothing. Shielding of the body from infra-red by an opaque object is, of course, the greatest protection. Occasionally flash burns are seen after explosions of gun powder in gun turrets, or other confined spaces and after explosion of steam boilers. There is some evidence to indicate that third degree burns are about twice as lethal per unit of surface area as are second degree burns.

In atomic explosions, intense infra-red radiation is liberated, and the lethal effects of burns are coupled with those of the ionizing radiations. In experimental animals, the lethal effect of burns is intensified by sublethal doses of ionizing radiation to the body. Burns which are seen after atomic blasts are those produced by heat and not by ionizing radiation, since a quantum of ionizing radiation sufficient to cause a burn would be lethal if exposure were given to the entire body surface. Shielding of the liver and spleen does offer some protection in total body radiation. One only sees local ionizing radiation burns after carefully shielded X-ray therapy, or, in the fall-out from atomic bombs. Vigilance for complications should be maintained.

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The Nature of the Thermal Injury

As the temperature of tissue is slowly raised, the chemical reaction rates will increase. The reaction rate doubles itself for each 10° C. rise in temperature (van't Hoff's law). The basal metabolic rate increases seven per cent for each degree Fahrenheit rise in temperature. Prolonged exposure to heat can produce cellular anoxia and death. In burns the heat is usually of shorter duration and more intense. When the temperature is raised further, protein is denatured and the cell is injured or destroyed completely. If tissue is immersed in water with a temperature over 50° C., exposure for one minute will produce irreversible damage. As the duration is increased it is natural that the damage becomes more widespread. As the extent of tissue destruction increases the burn becomes more lethal. Not only is the skin injured and destroyed, but blood vessels in the area are also injured and destroyed, and the selective permeability of capillaries is lost allowing protein to escape into the interstitial spaces. At temperatures of 45° C., the increased lymph flow does not contain appreciable quantities of protein, but at 50° C., the lymph fluid becomes highly proteinized. The protein does disappear with the cessation of heat, but above 50° C., the protein concentrations of the lymph gradually approach those of the plasma, and there is no return upon cessation of heat. With mild heat cellular disintegration is seen to occur, but when the heat is intense, heat coagulation occurs. In any case, there is always a thin shell of cellular disintegration which surrounds the completely coagulated tissue. Cellular disintegration is evidenced by the appearance of nucleic acid and basophilic granules in burn blister fluid.

Injury to Red Blood Cells

Since there is thermal injury to the vessels, the formed elements within them must also be injured. There is a sizable red cell destruction in extensive burns, which has been estimated to be between seven and ten per cent. Thus red cell hemolysis not infrequently causes hemoglobinemia and even hemoglobinuria. When blood is drawn for hematocrit or other chemical determinations, one should always look to see whether the plasma is pink. As a rule the majority of damaged erythrocytes are removed from the circulation in eighteen hours.

However, continued erythrocyte destruction goes on slowly for some time. This shows that some cells are mildly injured and are phagocytized more readily than they would have been normally. A change occurs at the red cell surface which allows it to stick to the reticulo-endothelial cells against which it bumps in its intravascular travels. A similar change at the cell surface occurs with malarial parasitization and coating of the cell in erythroblastosis. This change in the red cell envelope is fundamental since the reticuloendothelial cell has no way of knowing which cell is old or injured. It is the red cell itself which notifies the reticuloendothelial cell by developing a change at the surface which causes itself to stick and to be phagocytized by reticuloendothelial cells, especially those of the spleen.

For the above reasons, it is important to prevent coating of the cells with A and B isoagglutinins which may occur from the use of pooled plasma. This can be accomplished by adding Witebsky's substance to the plasma which neutralizes the agglutinins. It should be pointed out that this danger does not exist in patients with type 0 blood since their cells contain no isoagglutinins with which the serum isoagglutinins can react. A hemolytic anemia can occur in type A, B, or AB recipients from the transfusion of plasma from type 0 donors. This occurs even though intravascular agglutination does not occur.

Decreased Red Cell Formation

It is not merely the increased red cell destruction in burns which causes anemia, but at the same time there is a marked depression of red cell formation. This can be demonstrated with radioactive iron and isotopic glycine studies. These studies reveal that the depression of red cell formation is similar to the anemia of chronic infection, where a similar depression of iron utilization occurs. There is a fundamental disturbance in porphyrin metabolism and abnormal protoporphyrins are found in the newly

formed red cells. The resulting anemia must be treated with blood transfusions. Unfortunately, the red cell count and hemoglobin determinations cannot be fully relied upon as an indication for transfusion. When blood volume studies are done, a reduced blood volume is invariably found. The body compensates for this reduced blood volume, and there are no blood pressure or other changes which will denote its presence. This reduction in blood volume has been called "chronic shock." Chronic shock invariably develops during burn convalescence and even though the concentrations of hemoglobin and red cells may approach normal values, a deficit still exists. The best results are obtained if blood replacement is calculated on predicted normal volumes rather than on the actual volumes found. Since blood volume determinations are usually not performed during the clinical management of burns, it must be remembered that the burned patient will require more blood than is evident from blood counts.

Chronic shock is an important clinical entity since these patients already have a reduced blood volume, and a small blood loss will therefore throw the patient into acute shock. The patient will not tolerate the blood losses which may occur in changing dressings. During the removal of dressings from granulating surfaces blood losses up to 500 cc. have been measured. The blood losses during skin grafting procedures on burns are often in excess of 1000 cc. If one is to err, it is perhaps best to err on the side of over-transfusion, since the only hazard which will occur with over-transfusion is hemochromatosis. It should be pointed out that no cases of hemochromatosis have as yet been reported from over-transfusion.

Electrolyte Requirements

As the cells die, the selective permeability of the cell membrane is lost. The vascular tree now communicates with the tremendous dead space which formerly had a composition different from that of extracellular fluid, but which will now take on a composition identical to extracellular fluid. Thiocyanate determinations show an increase in the extracellular fluid space. In the normal human, the thiocyanate space is almost equal to the radiosodium space. In burns, the radiosodium space expands out of proportion to the thiocyanate space, since sodium gains access to injured cells where formerly it was excluded. When the selective permeability is lost, potassium is also released by the cell. The blood levels of potassium may rise, and although these levels are not lethal for normal animals, they might be lethal for the more potassium sensitive animal in shock. It is essential to preserve urinary function so the potassium may be excreted. Red cell destruction contributes to the potassium load.

Since sodium disappears into cells from which it was formerly excluded, an acute sodium deficit ensues. This serious need for sodium was not fully appreciated until recently. Prior to 1940, the emphasis was placed primarily on the infusion of plasma. As a matter of fact, saline infusions were even considered deleterious since they were supposed to wash out increased quantities of serum proteins. The extent of shift of sodium into the burned area of experimental animals is of magnitude sufficient to produce vascular collapse in normal animals from acute sodium depletion. The release of intracellular potassium and the sudden loss of extracellular sodium into the burned tissue produces an electrolyte pattern which is quite similar to that seen in acute adrenal insufficiency. This striking similarity between burn shock, and shock in adrenalectomized animals, attracted the attention of early investigators. Any equivocal beneficial action which resulted from the administration of D.C.A., A.C.T.H. or Cortisone probably resulted from a slightly favorable effect which these hormones might briefly exert in such a situation. There is no evidence that A.C.T.H., Cortisone or any other steroid hormone enhances survival in burned animals. These substances should not be used clinically to combat burn shock. The electrolyte imbalance should be corrected by the administration of sodium chloride, sodium lactate, sodium succinate, or any other sodium containing compound. In radiosodium experiments, the administered sodium accumulates quite rapidly in the burned area.

The need for sodium is rapid and the effects of sodium loss are seen very early in the course of burn therapy. The requirement tapers off after three or four hours, and by the end of forty-eight hours, there is no increased need. Edema formation reaches its maximum by forty-eight hours. Radiosodium determinations show that exchange of sodium in the injured pool is rapid, and equilibration with extracellular fluid sodium occurs in one or two hours. This is in contrast to plasma protein, which equilibrates slowly. It also points out that after forty-eight hours, the situation with regard to sodium is reversed, and the sodium will start to leave the burn site quite rapidly.

The intense need for sodium is reflected by the absence of sodium in the urine. There is maximal retention of sodium which is primarily on a hormonal basis. The glomerular filtration rate falls precipitously during burn shock and produces an oliguria. With the marked diminution in filtration rate, sodium will be absent in the urine. Complete tubular reabsorption from the scanty filtrate will occur. Even when the hemodynamic aspects are maintained, sodium reabsorption by the tubules is maximal because of a hormonal effect. This sodium retention outlasts the need for sodium, and by the third or fourth day, when sodium starts to be released from the burn site, dietary sodium must be restricted.

Fluid Loss

In addition to the need for sodium, there is also a need for water. The injured tissue is slightly overhydrated, but the main need for fluid is caused by the extensive edema in the burn site. Vascular injury allows the escape of plasma proteins, and it is the high protein concentration of the edema fluid which prevents the return of the fluid into the intravascular space. It is the extensive loss of fluid which reduces the circulating fluid volume and precipitates burn shock. Studies of fluid loss in fatal trauma give values for fluid loss in the region from four to five per cent. This value is almost the same as that of fatal blood loss which is in the region of four and one-half to five per cent of body weight. A toxic factor is probably also present in addition to the fluid loss. Because of the intense fluid loss, a relative dehydration of the normal tissue elsewhere in the body rapidly ensues. For that reason, one of the first clinical signs of burn shock is thirst. The avidity for fluid and the edema extends some distance into seemingly normal tissue. One of the aims of therapy is to satisfy the requirements of the burn pool for salt, water and protein. When this requirement has been fully satisfied, there will be no further loss into the burned area, and the hemodynamics can be maintained.

Protein Loss

It is evident from the aforegoing discussion, that not only fluid and electrolytes are lost, but protein is also lost into the area of injury. This problem of plasma protein loss was at one time thought to be due to a generalized increase in capillary permeability. When the plasma protein molecules were marked by a radioactive tag, it was found that although plasma albumin appeared in high concentrations in the lymph of the burned extremity, it did not appear in the lymph of normal extremities until terminal shock appeared. That there is no generalized increase in capillary permeability has been confirmed in many studies using radioactive plasma proteins. Nevertheless, all of the protein which is lost does not accumulate at the burn site. Much of the plasma protein loss is brought about by the metabolism of protein to urea. The increased catabolism of protein is initiated via the pituitary-adrenal relationship. The alarm reaction causes liberation of A.C.T.H. from the pituitary. This in turn results in the secretion of hydroxycortisone from the adrenal cortex. Hydrocortisone facilitates the conversion of protein to glucose (gluconeogenesis). The end product of the protein metabolism is urea.

Although hypoproteinemia rarely develops during the acute course of burn shock, it can occur if extensive infusion of non-colloid substances are given. The loss at the burn site is practically pure plasma.

Infused radioactive plasma protein rapidly accumulates in the area of injury, but this protein does not readily exchange

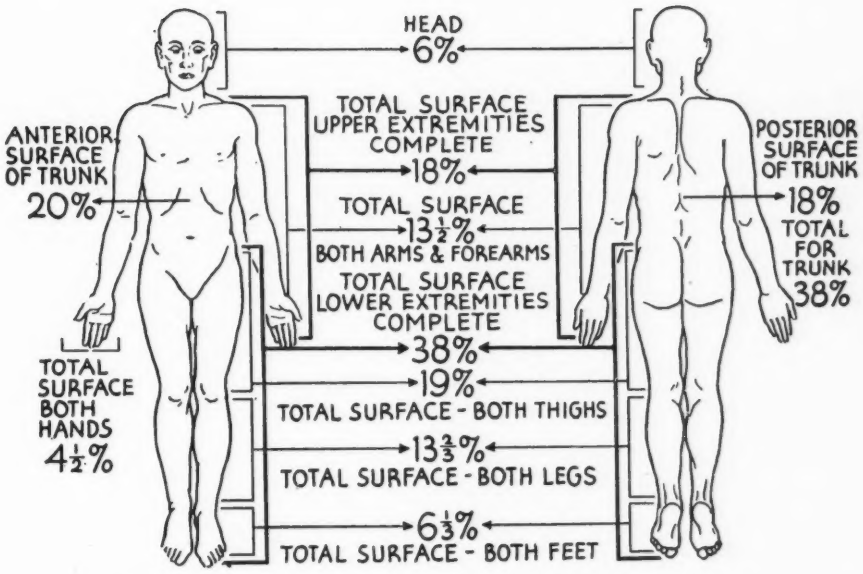

Figure I: Berkow's method of estimating the extent of surface area burned. (From Seeger, The Treatment of Burns, Lewis Practice of Surgery.)

with plasma proteins as was observed in the case of sodium. Equilibrium of the burned protein pool with plasma protein pool is slow and requires at least eighteen hours. In essence, the protein is sequestered from the circulation. After the burn pool is saturated with fluid and protein, less protein will be lost. For this reason, the hemodynamic effect of infused plasma is always greater when infused late in burns, since the rate of loss is less rapid. Like the salt requirement, the plasma requirement is high initially and tapers off continuously.

The plasma loss, to a great extent, is linearly related to the percentage of surface area which is burned. Burns of less than fifteen per cent in adults and eight per cent in children do not usually show signs of shock. The extent of surface area involved is also related to the mortality. Mortality of fifty per cent can be expected in patients properly treated with plasma, in burns involving fifty per cent of the body surface. It is essential that the exact extent of surface area burned be accurately measured at the time of admission to a hospital.

Surface Area

Estimation of surface area cannot be done haphazardly, but some exact method must be used to estimate the percent of body burned. Wallace's "Rule of Nine" is popular since it is easily remembered. It makes use of the fact that the surface area of various parts of the body are multiples of nine. The head is nine per cent; each upper extremity is nine per cent; the anterior trunk is eighteen per cent; the posterior trunk is eighteen per cent, and each lower extremity is eighteen per cent. So that the total will be 100 per cent, one per cent is added for the neck. Another method for estimating the extent of a burned area is Berkow's method. (Fig. 1.) This latter is the most exact method and should be used whenever possible. It is the surface area figures, which allows for comparison of therapy, to see which type of therapy is the most successful. When over seventy per cent of the body surface is involved the mortality rate is over ninety per cent.

Burn Shock

It is the rapid loss of circulating blood volume which precipitates burn shock.

The hemodynamic aspects of shock are well known: the pulse is weak and rapid; the venous pressure falls; the blood pressure falls; and, related to this generalized deterioration of the circulation, tissue anoxia occurs. This is manifest by excitement, restlessness and rapid shallow respirations. The skin is usually cold and moist. Although pallor is usual, occasionally the skin color will have a blotchy cyanotic appearance. The mouth is dry and the patient complains of thirst. There is a complete suppression of urinary output.

The biochemical changes which accompany shock are of a serious nature and irreversible damage will occur if the tissue anoxia persists. The sluggish circulation of blood through the tissues produces widening of the arterial-venous oxygen difference, since the tissues extract the maximum amount of oxygen from the sluggishly circulating blood. Although all tissues suffer from anoxia, it is in the liver where the most lethal anoxia occurs. Most of the oxygen is removed from the blood by the intestines, decreasing the oxygen supply which the liver normally derives from the portal vein blood. Although three-fourths of the blood flow of the liver comes from the portal vein, the liver only derives fifty per cent of its oxygen requirement from this source. The precipitous fall in blood pressure also reduces the blood supply from the hepatic artery. In experimental animals, occlusion of the hepatic artery is fatal and produces a syndrome hemodynamically and biochemically similar to shock. Vasodepressor material is manufactured by ischemic liver, muscle, and spleen. This vasodepressor material is inactivated by the normal liver. Inactivation does not occur in the hypoxic liver, and after liver anoxia has existed for some time, the liver does not regain its capacity to inactivate this vasodepressor material. Some of the vasodepressor and toxic material originate from absorption of toxic material which is produced by the intestinal bacterial flora. In experimental animals, sterilization of the G.I. tract increases the resistance to hemorrhagic shock.

The failure of liver metabolism brings about a situation which is comparable to that observed in hepatectomized animals. There is an accumulation of lactic acid in the blood, and a severe acidosis results. The liver is incapable of metabolizing the lactic acid which is produced by ischemic muscle. The conversion of ammonia to urea is also impaired. A return of liver function is possible if the circulating dynamics are restored promptly, but prolonged anoxia leads to irreversible damage.

In burn shock, the loss of plasma produces a hemoconcentration. The hematocrit concentrations rise and causes the blood to become more viscid. This increases peripheral resistance and tends to further slow the sluggish circulation. There is extensive peripheral trapping of erythrocytes in the capillary loops. Many of the trapped erythrocytes are returned to the circulation after plasma infusion. In addition, intravascular clumping of red cells has been described by Kneisly. This intravascular clumping has been described as "sludged blood." Burns are one type of injury which, understandably, can produce extensive sludging of the blood. This will lead to additional trapping of erythrocytes in the capillaries.

Therapeutic Implications

Since the plasma and fluid loss into the tissues causes edema and swelling, it is natural that application of pressure (or a restrictive dressing such as a plaster cast which is applied before swelling occurs) would reduce the extent of swelling. Pressure dressings reduce burn mortality in experimental animals. Elastic bandages have become quite popular clinically, for the closed method of burn therapy.

The extensive fluid and electrolyte loss makes it mandatory that large quantities of electrolyte solutions be given in addition to plasma. The fluid and electrolytes are usually given by mouth, but they may be infused. Berman has suggested that the burn site be infiltrated with normal saline to rapidly satisfy the burn pool's requirement for salt and water. This procedure may have merit in some situations, but as a rule the oral route will prove satisfactory in most cases. Fox administered ten to fifteen

per cent of the body weight in the form of one-sixth molar sodium lactate in a twenty-four hour period (seven to ten liters). Even though these patients were not given plasma they responded well. This quantity of fluid is slightly excessive if plasma is used, and it seems doubtful whether more than four or five liters of fluid are ever required when combined therapy is employed.

The author favors Moyer's suggestion that sodium bicarbonate salt solution be used. Moyer gives a solution containing four grams of sodium chloride and one and one-half to two grams of sodium bicarbonate to the liter. This solution tends to combat aforementioned acidosis which is always a serious complication of burn shock. Evans does not employ the oral route, but infuses a quantity of sodium chloride equal to that of plasma.

A number of attempts have been made to relate plasma loss to some mathematical expression. This has resulted in many complex formulae for calculating the plasma dosage required. When a comparison of methods is made there seems to be little virtue in the complex formulae over the simple. Since formulae are merely guides for plasma administration rather than rules, it is foolhardy to place undue emphasis on them.

One group of formulae is based on the observation that plasma loss tends to produce hemoconcentration and hypoproteinemia. The other group empirically relates the degree of plasma loss to the extent of burned surface. The relationships between hemoconcentration and plasma requirement may be briefly summarized as follows:

1. Give 100 cc. of plasma for each point the hematocrit is above the normal of forty-five.
2. Give fifty cc. of plasma for each point the hemoglobin exceeds the normal of 100 per cent.
3. Give 100 cc. of plasma for every 100,000 the red cell count exceeds the normal of 5,000,000.
4. Give 150 cc. of plasma for every specific gravity increase in .001 over the normal blood specific gravity which is 1.060.

It is my feeling that blood counts and hematocrit determination are best utilized to determine the adequacy of burn therapy, rather than attempting to utilize this information to determine total plasma dosage. If the hematocrit can be kept within normal limits, the plasma therapy is usually adequate.

The first aid formula is one of the best for routine clinical usage. According to this formula, fifty cc. of plasma is given for each per cent of body surface burned. On the basis of studies of protein loss, some have recommended that this amount be increased to sixty cc. However, Evans points out that the use of formulae based on a percentage of body surface burned may be hazardous when applied to burns involving fifty per cent of the body surface, and in people over fifty years of age. Evans estimates the plasma loss as follows: plasma requirement = weight in kilograms × per cent of burns × 1 cc. This estimate is more sensible than merely basing plasma requirement on the percentage of body surface burned, since large people will naturally have more surface area and smaller people may be overtreated.

Because the plasma is lost most rapidly in the early phases of the burn, the plasma dosage should be divided as follows:

1/3 in the first two hours,
1/3 in the subsequent four hours, and
1/3 in the subsequent six hours.

The highest survival rates have thus far been obtained when a combination of electrolytes, plasma and blood are used. Blood should be substituted for at least 1000 cc. of the plasma requirement.

Where plasma is not available dextran should be used. This substitute is the least toxic of the plasma substitutes and does not have antigenicity or other harmful side effects. It is a polysaccharide which is slowly metabolized and excreted by the body; even so, it depresses plasma proteins during the interval that it circulates. This is not altogether undesirable since plasma proteins are slowly replaced as the dextran is slowly removed from the plasma. In experimental burns dextran is not as effective as plasma, but the differences are not

striking. At the present time it seems wisest to reserve the use of dextran for mass casualties when the supply of plasma would be inadequate.

If therapy is vigorous and adequate urinary output will be maintained. One should try to see that the urinary output is kept at about thirty-five cc. during the first hour. The initially passed urine should be examined for hemoglobin. An adequately sustained urinary output is one of the best indications that normal hemodynamics are being maintained. In any situation where shock has occurred, anuria and complete renal shutdown may occur as a complication. Shock is a more frequent cause of anuria than is the transfusion of incompatible blood.

There is no contraindication to the proper sedation of burned patients. The reduction in body activity will reduce the metabolism rate and have beneficial action beyond this value of sedation. Although newer derivatives have been suggested, morphine is still the drug of choice for most clinicians.

Local Therapy

There is considerable agreement about the definitive general therapy of burns. With regard to the procedure for the care of the burn site itself, there is widespread difference of opinion. In general the therapy can be divided into two categories, open and closed treatment. Some years back tannic acid was utilized in an attempt to reduce the surface exudation. Tannic acid therapy has been abandoned since it has been implicated in causing liver damage. In addition, second degree burns are often converted into third degree burns. Often, a problem presented itself in the removal of the eschar and occasionally pus collected underneath the eschar, which in itself impeded drainage.

Many substances have been suggested for the local therapy of burns with the intent of reducing bacterial contamination and stimulating epithelization. It is impossible to keep a burn wound sterile. Also, there have been no materials which when applied locally or systemically will promote epithelization. In the Coconut Grove fire, great success was achieved by merely wrapping the burns up without attempting to even cleanse them. Blister fluid has been shown to be sterile and blisters should not be opened without a specific reason. In the closed therapy, the wound is usually dressed with vasolinized gauze and pressure exerted on the burn with a rubberized elastic bandage.

There are some who favor the so called open treatment of burns. In this therapy, the wounds are left open to the air and no dressing is applied. These advocates claim that a thin crust of plasma coagulum forms over the wound site and protects it. They claim that there is less burn toxemia with open as compared to closed therapy.

A combination of both types of therapy is probably best. Initially, closed therapy can be employed and later in the course of treatment, if there is fever and evidence of absorption of bacterial products, tub soaks containing sodium chlorides can be used to advantage.

Even though antibiotics are used extensively, the burn site becomes heavily contaminated with staphylococci. It is interesting that therapy with penicillin has prevented the occurrence of hemolytic streptococci in burn wounds, but not staphylococci. A frequent contaminant is Aerobacter aerogenes. (B. pyocyaneus). This organism gives the dressings a typical "barnyard" odor. The only importance of bacterial surface contamination is that some bacterial products prevent successful takes of skin grafts. Pyocyaneus produces a proteolytic enzyme which attacks the graft itself and prevents the establishment of a blood supply. The ammonium which is produced also impedes a successful take. Hemolytic streptococci produce fibrinolysin which dissolves fibrin. It is fibrin which primarily attaches the graft to the recipient site. The products of some staphylococci also interfere with takes of skin grafts. It is wise to eliminate those specific organisms which jeopardize the take of skin grafts. A one per cent acetic acid solution has been used for pyocyaneous, but it is not as effective as Polymyxin.

It is obvious that early skin grafting is the ideal therapy for burns. The burn site must be prepared before successful grafting can be done. This involves the

removal of all necrotic debris, so that a clean granulating base is offered for attachment of the graft. Preparation of the wound may involve mechanical removal of dead skin with a scissor and forceps. Various proteolytic enzymes may be used for wound debridement. The theory behind the use of these substances is that the enzymes can differentiate between dead and live tissue more readily than can the eye. Viable tissue is not readily liquified. The enzymes used for these purposes are trypsin, papain and streptolysin. Crystalline trypsin is commercially available and nontoxic. It can be injected beneath an eschar, or can be applied locally in the form of a jelly. Its great disadvantage lies in the fact that the body builds up high antitryptic titers which render subsequent applications less effective. Streptolysin is pyrogenic and also becomes less effective on subsequent applications.

Nutritional Problems

Every burn involving more than ten per cent of the surface area produces a nutritional problem. Nitrogen balance is difficult to maintain because of excessive protein loss. Initially, the stress mechanism produces a loss of protein by gluconeogenesis. Later on, there is an extensive loss of protein from burn exudates. Co Tui has found the nitrogen loss to be as great as .42 milligrams per square centimeter of body area per day. If one-half the body surface was burned in a normal size man this would involve an extent of 90,050 square centimeters. The loss would be equivalent to 124 grams of protein or 2000cc. of plasma. If it takes three grams of protein to form one gram of plasma, the loss would require the ingestion of 1800 grams (over three pounds) of meat a day, merely to replenish the loss. In addition, fever raises the metabolic rate seven per cent for each degree rise in temperature. Chronic infection in the burn site produces anorexia so that the patient cannot be relied upon to take sufficient food even though it is offered. Maintenance of the severely burned patient requires a minimum of two or three grams of protein, and at least sixty calories per kilogram of body weight. When a protein deficit occurs these figures must be revised upward.

The only way to meet the serious nutritional requirements of the burned patient is by force feeding day and night. A small polyethylene catheter should be placed in the stomach. Day and night the patient is given high protein, high calorie liquid foods through the tube. A fat emulsion and a mixture of protein and carbohydrates can be made up from the commercial preparations or merely by mixing one-half milk and one-half cream and adding additional dry milk powder to the mixture. The protein hydrolysates may also be used, but they have no benefit over native proteins. Babies' formulae using evaporated milk or other products are ideal.

It should be pointed out that the late mortality in excessive body burns has not changed and is still high. Nutritional deterioration is one cause for the high late mortality in burns.

Complication of Burns

Occasionally duodenal ulcers are seen as complications in the convalescence of burns. These ulcers are typical peptic ulcers and are called "Curling's Ulcer". Ulcers have been found to develop during cortisone and A.C.T.H. therapy. Existing ulcers sometimes perforate or hemorrhage during a course of hormone therapy. It has been shown that A.C.T.H. and cortisone increases the secretion of hydrochloric acid and may also reduce the resistance of the duodenal mucosa to ulceration. Any stressful situation which calls forth the endogenous secretion of A.C.T.H. may produce ulcers. Such ulcers have been described after abdominal operations, fractures, and a variety of other trauma. More recently, they have been given the descriptive name of "stress ulcers". If patients are treated around the clock with high protein feedings, the danger of peptic ulcer development is slight. Symptoms, such as heart burn and epigastric pain, are warning signals.

Anuria can result from shock, hypotension or hemoglobinuria. The renal lesion which produces the oliguria or anuria has been designated as acute lower nephron nephrosis. This complication is most often fatal. The adminis-

tration of fluid must be restricted to the actual losses. Insensible water loss approximates 1500 cc. per day, with an additional 200 cc. required for each degree rise in temperature. As a rule, a high carbohydrate protein free diet is given to keep protein catabolism at a minimum.

Occasionally pulmonary burns or respiratory injury occur. This is especially frequent with steam explosions which may produce pulmonary edema and death. One should always keep in mind the presence of respiratory tract involvement. This was not an uncommon cause of death in the Coconut Grove fire.

Contractures may develop about a joint if one is not careful to prevent them. Children with abdominal and thigh burns will frequently keep the thighs flexed on the abdomen. It may be necessary to splint the knee and ankle in some cases. Pressure sores should also be guarded against by preparing padding of the heel, Achilles tendon and bony prominences.

SUMMARY

The essential features in burn therapy may be divided into the initial and late therapy. The proper initial therapy depends upon the administration of electrolytic fluids, plasma, and blood. The salt water solution may be given by mouth or infused. Approximately fifty cc. of plasma should be given for each per cent of body surface after this has been determined by "Rule of Nine" or Berkow's Method. Blood, in the amount of 1000 cc., should be given initially to severe burns, and transfusions given periodically during the convalscent period. Salt and water, equal in quantity to the administered plasma, should be given via the oral route. Local burn therapy consists of the application of vaseline gauze, and pressure applied with an elastic bandage. Sedation is given on admission to the hospital.

The late therapy of burns involves maintenance of nutritional status by surfeit feeding and preparation of the burn site for skin grafting. Systemic penicillin prevents contamination by hemolytic streptococci. Debridement of the burn wound is performed mechanically or chemically. One must anticipate complications.

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OCULAR INJURIES

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The purpose of this article is to acquaint physicians, other than eye specialists, with the basic techniques of initial diagnosis and treatment of eye injuries. There have been several new contributions in the ophthalmic literature concerning trauma of the eye during the past two years[1]. The increasing mechanization of our society, coupled with the ever increasing use of materials which can traumatize the eye, necessitates that everyone in the medical profession be acquainted with the early diagnosis and treatment of eye injuries. The senior medical student and the intern especially, should be aware of the correct methods of examining these patients. A patient that has had a lid laceration sutured in the accident room may appear in the eye clinic several days later with a severe hemorrhage in the anterior chamber of the eye or with a scleral laceration that may have been overlooked at the time of the initial examination. Several articles, to which reference will be made, clearly demonstrate that proper initial management by the non-ophthalmic physician, who first sees the case, and later by the ophthalmic surgeon will result in the saving of useful vision in many injured eyes.

Injuries to the eyes are common, although the eyes may be said to be in a position of relative safety, protected as they are by the bony orbital rim, the nose, and a bed of orbital fat and elastic tissue. The blink reflex, the head-turning reflex, and the tear-mechanism are sometimes insufficient protection against injury.

Duke-Elder[2], in reporting the statistical incidence of ocular trauma, found in 1949, that thirty per cent of the eye admissions to a hospital in an industrialized part of London were due to injury. Bellows[3] reported twelve to seventeen per cent of all injuries in World War II occurred to the eye. In children alone, Werner[4] reported an incidence of eighteen per cent of all hospital eye cases in a five year study. At Cook County Hospital approximately fifteen to twenty per cent of the eye admissions, including both in and out-patients, are the result of injury. Articles by Knap[5] and Welczek[6] show that males receive eighty to ninety per cent of ocular injuries probably due to their predominance in industry. However, if industrial accidents are not considered, it has been shown that fifty per cent of eye injuries occur in children. This should be of special interest to the pediatrician.

DIAGNOSIS AND TREATMENT BY THE NON-OPHTHALMOLOGIST

An accurate history of an eye injury may be of great value in determining the type of injury and the extent of damage. Information concerning foreign bodies should include, in addition to the nature of the missile, the character, and force, and direction of entrance, to aid in the localization and treatment of the injury. As the eye may be relatively insensitive to a number of materials, an accurate history may not be obtained. In these cases, the physician must then rely completely on the physical examination.

The initial examination of the eyes should be performed with extreme care. Pressure on an injured eye should be avoided at all costs. The dangers of vitreous loss, intraocular hemorrhage, and retinal detachment are all increased by careless movement, either by the patient or the attending physician. When possible, the eyes are best examined with the patient in the upright position. The lids should be separated and the conjunctiva, cornea, and sclera carefully examined. When the lids are tightly closed due to spasm or swelling, it is advisable to inject the lids with a local anesthetic agent, such as four per cent Procaine, and separate them with lid hooks or sutures placed in the lid mar-

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gins. Abrasions, ulcerations, or foreign bodies of the cornea can be seen by placing a drop of sterile two per cent sodium flourescein on the cornea. Those areas in which the epithelial layer has been destroyed will stain a brilliant green. Any foreign material which may be present will stand out in sharp contrast, thus making its removal easier. The bulbar conjunctiva, because of its mobility, may appear intact in penetrating wounds of the eye and thereby produce confusion in detecting underlying injuries. A wound in the sclera may be diagnosed by the presence of pigmented uveal tissue penetrating the sclera.

Lacerations of the cornea, if small, may be overlooked. However, the presence of lacerations may be suspected by a flattening of the anterior chamber resulting from leakage of aqueous, and/or an irregular pupil because of an adhesion, incarceration, or prolapse of iris tissue into the corneal wound. The presence of a hyphema is noted when blood is seen in the anterior chamber. A tremulousness of the iris indicates that the lens has been partially or completely dislocated. Ophthalmoscopic examination may reveal a loss of the red fundus reflex as a result of hemorrhage into the vitreous. A retinal detachment or an increase in the intra-ocular pressure (secondary glaucoma) must always be suspected, although it may be difficult for the non-specialist to diagnose those complications which are likely to occur in concussion type and penetrating injuries.

Other findings which are helpful in the diagnosis of ocular injuries are:

1. *Diplopia*: Indicates an injury to either the extra-ocular muscles or their nerves.
2. *Enophthalmos*: Probable fracture of the floor of the orbit.
3. *Orbital emphysema*: Seen in fractures of the para-nasal sinuses.
4. *Loss of vision*: If present in the presence of a good red fundus reflex one must suspect damage to the optic nerve. Avulsion of the optic nerve and fracture of the apex of the orbit are two causes for this condition.

The attending physician should record all physical findings as they may be of great assistance to the ophthalmologist. It is most important that *both* eyes should be observed in every instance of injury.

Initial therapy by the non-specialist should start with putting the patient at complete bed rest so as to avoid further trauma to the eyes. The most important exception is in the case of chemical injuries. These should be thoroughly irrigated and carefully flushed immediately. Pain should be alleviated with systemic medications. Tetanus antitoxin should be administered if there is no history of immunization, and the systemic administration of a wide-spectrum antibiotic begun. The non-specialist *should not* place any medication in the eyes. The reason for this is that many of the commonly used ophthalmic preparations are harmful in certain types of injuries. Atropine may produce an increase in intra-ocular hemorrhage or may aggragate a prolapse of the iris. Antibiotic cintments, in the presence of a corneal laceration, may enter the eye, producing a severe systemic reaction. Local anesthetics have been shown to impede corneal metabolism and mitosis and, therefore, are usually contraindicated. After relieving the general symptoms, the patient should be placed in a recumbent position and a *firm* binocular patch applied which will prevent blinking of the eyelids. Referral should then be made for definitive diagnosis and treatment.

DIAGNOSIS AND TREATMENT BY THE OPHTHALMOLOGIST

The ophthalmologist has at his disposal special diagnostic equipment and tests which are of great value in the examination of the eye. These are used in cases of ocular injury, for definitive diagnosis and treatment. The visual acuity of each eye is first accurately obtained. A gross examination of each eye is then done. Afterward each eye may be examined by some or all of the following procedures:

1. *Biomicroscopy and Gonioscopy*: The biomiscroscope (slit-lamp) gives the examiner an opportunity to view the ocular structures under high magnification

and with intense illumination. Some of the common injuries seen with this instrument are small corneal and scleral lacerations, uveitis, tears in the iris sphincter or iris root (iridodialysis), lens opacities or dislocations, vitreous hemorrhage, and, by means of special attachment, retinal pathology.

Gonioscopy consists of placing a special contact lens on the cornea which permits the examiner to scrutinize the structures located in the angle of the eye formed by the junction of the cornea and iris. Foreign bodies, not seen in earlier examinations, may, in this manner, be visualized in the angle of the eye.

2. *Tonometry*: The determination of the intra-ocular pressure is routine in all ocular examinations. It is especially important in deciding upon the treatment of certain ocular injuries, particularly hyphema. The onset of secondary glaucoma is frequently an indication for surgery. However, the tonometer should not be placed on a severely injured cornea or used in the presence of infection. Palpation of the eyeball for increased tension must suffice in these cases.

3. *Visual Fields*: Field studies are most important in estimating the integrity of the retina and the optic nerve pathways to the occipital region of the brain. In addition, it is invaluable in following lesions of the macula and retinal detachments.

4. *Ophthalmoscopy*: The ophthalmoscope may be used in the diagnosis of trauma to the lens, vitreous and retina. The cornea and lens must be transparent to permit successful examination of the posterior segment of the eye. Severe concussions frequently produce retinal edema, retinal hemorrhage, macula injury, or retinal detachment.

5. *X-Ray*: Roentgenograms of the eye should be taken early, in all cases of possible intra-ocular or intra-orbital foreign bodies. Fractures of the orbit or nasal sinuses should always be suspected. Bilateral roentgenograms are often helpful for comparative studies. Frontal and lateral views of the orbit, the orbital foramina, and the nasal sinuses, as well as a lateral film of the skull are ordered. If a foreign body is noted, the patent is re-examined, this time using special radiologic technique for localization (*i.e.*, Sweet's method)[7]. Most foreign bodies, in this manner, can be accurately localized, allowing the surgeon to remove them with a minimum of additional trauma.

TREATMENT

The treatment of ocular injuries depends on the etiology and extent of damage. Five of the most frequent causes of injury will be discussed with reference to the most recent advances in their treatment.

Burns of the Eye

The most common causes of burns are those due to thermal and chemical agents. Each will be considered separately.

a. *Thermal injuries*:

A rather complete report on the management of thermal injuries has been published by Leahy[8]. As with all cases of burns, the patient's general condition must be thought of first. The presence of shock necessitates the administration of blood, plasma, intravenous fluids, and other emergency measures. The principle guiding the treatment of eye injuries is to restore the tissues as rapidly as the patient's condition permits, to prevent scarring.

In cases where there are full thickness burns of the eyelids, it may be advisable to resect all of the burned tissue and to place a full-thickness graft in the exposed area. If a contracture is to be prevented, intermarginal lid sutures must be placed at the time of surgery in order to immobilize the lids. This principle holds for burns of the conjunctiva as well, except that in this case, mucous membrane grafts are used.

The treatment of partial thickness burns, where viable epithelium remains, does not necessitate grafting. Local antiseptics, if applied, delay healing and, therefore, should not be used. A clean vaseline dressing, together with the sys-

temic administration of antibiotics usually is adequate.

The above measures, if performed early, will prevent contractures, symblepharon, entropion, or ectropion. The use of local cortisone has been shown to impede neo-vascularization and fibrous tissue formation and may be of value as an adjunct in the early treatment of burns[9].

Burns of the cornea may be superficial or deep. The superficial types can be treated as a corneal erosion. They usually heal without scarring. Severe corneal burns must be approached with much more care because of the frequency of scarring and vascularization. The local care consists of cortisone, atropine, and systemic antibiotics. A corneal transplant may be ultimately necessary. Clinical judgment will decide the therapy in the individual case.

The late treatment of thermal burns of the eyes frequently involves extensive plastic procedures. This subject is well covered in an article by Spaeth[10].

b. *Chemical Injuries*:

The treatment of chemical injuries has been the subject of much discussion in recent years. An understanding of the action of these agents on the eye is important in explaining the pathogenesis of these injuries. Duke-Elder[11] describes three factors which determine the extent and type of damage following a chemical injury. They are:

1. *Intimacy and Duration of Contact of Agent*: Materials which stimulate the nose and corneal nerves, even in small concentrations, produce early lacrimation and blepharospasm which serve to protect the eye from serious injury. Some materials, such as mustard gas and hydrogen sulfide, do not produce stimulation and the patient who is exposed, therefore, does not receive any warning. This can account for the severe cases of keratitis which these chemicals will produce. The early and thorough irrigation and flushing of the eyes exposed to chemicals is, therefore, *most* important.

2. *Physical Properties of the Agent*: Two physical properties are important in chemical injuries. One is the solubility of the chemical and the other is its degree of penetrability. More soluble materials produce greater irritation to the eyes than the less soluble ones. The penetrability is, however, the most important factor. Swan and White[12] and later Cogan[13] showed that water soluble materials penetrate the epithelium of cornea with difficulty but pass through the stroma freely. This explains why acids, which are water soluble, produce little damage to an eye in which the epithelial layer is intact. Fat-soluble chemicals pass through the corneal epithelium freely but cannot penetrate the stroma. Alkalies, being fat-soluble, are much more damaging to the eyes than acids. The former are retained in the stroma producing marked cellular damage and coagulation necrosis. Substances, such as sulphur dioxide and mustard gas, which are both water and fat-soluble, are particularly damaging to the eyes.

3. *Chemical Properties of the Agent*: Chemicals produce their most damaging effect on the eyes by upsetting the chemical structure of the involved tissues. As a result, the pH of the tissues is disturbed, thus disrupting vital metabolism. Furthermore, these agents combine with reactive groups in the protein molecules (*i.e.*, the sulfhydryl group), to produce denaturation and breakdown of protein structure. Cellular oxidation is also depressed because of the immobilization of the necessary enzyme systems.

With these factors in mind, the rationale of treating cases of chemical injury to the eye becomes apparent. First, the eyes should be thoroughly irrigated with water or preferably saline. The use of neutralizing agents is definitely contraindicated in most cases since many of them are harmful to the eye. The next step is to prevent or control severe ocular inflammation, improve corneal metab-

olism, and prevent infection. Atropine and cortisone have been shown to be of great value in reducing an iritis. Cortisone may further control neo-vascularization. It was hoped that a new drug, five per cent Hydrosulphosol, advocated by Kuhn[14] and others would be helpful in improving corneal metabolism. They felt the administration of an excess of sulfhydryl groups to the injured eye would overcome the inactivating effect of the chemical agent. Reports by McLaughlin[15], Hardy[16], and Turtz and Horwich[17], however, have been discouraging and the drug has fallen into disuse. The administration of local and systemic antibiotics is urged to avoid secondary infection. The use of local antibiotics which are not used systemically is preferred.

The effects of chemicals on the conjunctiva is similar to that on the cornea with resultant necrosis, secondary scarring, and symblepharon formation. Early irrigation is most important and mucous membrane grafts, when necessary, will help alleviate this complication.

The ultimate use of corneal transplantation is indicated in those cases in which the corneal scars are central and in which there has been a minimal amount of neo-vascularization. Chemical injuries of the cornea are considered poor risks for transplantation because of the poor tissue metabolism.

Ocular Concussion and Traumatic Hyphema

Concussions involving the eye should always be considered as extremely dangerous because of the manifold types of injury that may occur. Hogan[18] emphasized the importance of careful examination and careful observation for several days with the patient at absolute bed rest. Hemorrhage into the anterior chamber is not uncommon following concussion. The treatment is bed rest and observation of the intraocular tension. If secondary glaucoma ensues, a paracentesis should be performed. The use of diamox in these cases is debatable since, by its action of reducing aqueous formation, it may also impede the resorption of blood. Mydiatics are to be avoided in the early treatment of hyphema as they may produce further bleeding and secondary glaucoma. The use of miotics, however, is not universally advised, although some ophthalmologists advocate its use routinely in hyphema. The use of cortisone and antibiotics is indicated to reduce inflammation and prevent infection. Other common complications resulting from concussions are scleral rupture, dislocation of the lens, vitreous hemorrhage, retinal edema (especially of the macula), retinal detachment, and avulsion of the optic nerve. In addition, the adjacent extraocular structures may be affected. Tears in the extraocular muscles, fractures of the orbital wall and damage to the third, fourth and sixth cranial nerves have been reported.

The treatment of each case is to be carefully considered. However, a ruptured eyeball is always repaired. Severely disrupted and hopelessly blind eyes are best enucleated to prevent the possibility of sympathetic ophthalmitis. The diagnosis of a typical case of sympathetic ophthalmitis is seldom difficult. A history of a penetrating wound or injury followed by inflammatory signs in the second eye should make one suspicious. If this occurs in the first few weeks, the diagnosis is almost certain. If the inflammation occurs after a much greater interval of time, other causes of uveitis must be considered and investigated.

The sympathogenic eye is one in which there has been a history of a penetrating wound or injury, followed by persistent inflammation and irritation. Examination reveals pericorneal injection, precipitates on the posterior corneal surface, a thickened, muddy looking iris with deposits and opacities on the anterior lens surface. The sympathetic eye may reveal signs of inflammation any time after the injury to the first eye. It is most commonly seen in the first six weeks. It is urged that routine examination of the opposite eye be performed, since it is common for objective signs of sympathetic ophthalmitis to appear before the patient is subjectively aware of any difficulty. The appearance of delicate ex-

udates in the aqueous and retrolental spaces and the presence of precipitates on the posterior surface of the cornea are characteristic findings. Optic neuritis and choroiditis have also been seen as primary findings.

The treatment of sympathetic ophthalmitis is primarily one of prevention. The use of modern surgical methods along with antibiotics and cortisone has greatly reduced the incidence of this condition. Winter[19] showed that once the signs of sympathetic ophthalmitis appear, enucleation is no longer indicated. He concluded after studying 257 cases of sympathetic ophthalmitis that: 1) Enucleation coincident with the onset of symptoms in the sympathizing eye does not improve the final vision available to the patient; 2) Cortisone and ACTH now provide a degree of control of sympathetic uveitis not previously attainable; 3) There is an error of about ten per cent in the diagnosis of sympathetic ophthalmitis.

Perforating Injuries of the Globe

Recent studies have shown that the use of new suture materials with exceptionally fine needles, the early use of antibiotics and cortisone have resulted in a reduction of the number of penetrating ocular injuries requiring enucleation. Duke and Schimek[20], and later Zekman and Lieberman[21] showed that only twenty to twenty-five per cent of reported injuries of this type were enucleated. Approximately one-third of the reported cases retained a vision of 20/40 or better.

The prime causes for enucleation are: 1) Irreparable loss of the intraocular contents; 2) Fulminating infection; 3) Phthisis; 4) Repeated destructive hemmorrhage. Each case must be considered individually. The initial examination frequently gives considerable information concerning the eventual prognosis. Complete skull and orbital roentgenograms should be obtained pre-operatively to rule out the presence of a retained foreign body. The early diagnosis and repair of perforating ocular injuries is important in the prevention of further complications, especially infection.

Lacerations of the Lids

A detailed discussion of the repair of lid lacerations cannot be included. Scheie[22], in 1952, summarized and illustrated some of the techniques used. It is important to stress that the lids are fragile structures with a definite anatomical structure. More harm can be done by suturing a lid improperly than by the injury itself. As example, a postoperative ectropion may result in a severe exposure keratitis while an entropion may produce corneal ulcerations secondary to contact with the eyelashes. In both instances blindness may ensue.

Adherence to certain basic principles will avoid these complications. If a lid laceration is first seen by someone inexperienced in this field it should *not* be repaired. Instead, the wound should be cleaned, covered with a clean vaseline dressing, and the administration of systemic antibiotics begun. A decision as to whether tetanus toxoid or anti-toxin is indicated, must be made in each case. Surgery should be performed within twenty-four hours by a competent surgeon. Because of the rich blood supply to the lids it is rather easy to get healing by first intention.

The most serious type of lid injuries are those which penetrate the lid margin. In these instances care must be taken to appose the two cut edges so that the so-called grey line separating the conjunctival-tarsal surface from the skin-obicularis surface are in perfect alignment. A tarsorrhaphy at the time of surgery may prevent late scarring and retraction of tissues.

PREVENTION OF OCULAR TRAUMA

It is recognized that the non-ophthalmic physician will be limited in the matter of treating ocular injuries. This is true because of the extremely delicate nature of the ocular structures. Furthermore, highly specialized instruments, which are not readily available, are frequently needed both for the diagnosis and the treatment. For this reason, special emphasis should be placed on the prevention of injuries to the eye. Certain specific precautions are of great value.

The industrial physician should carefully scrutinize his plant for possible sources of ocular trauma. Employees should be educated as to the hazards resulting from the improper handling of acids and alkalies. Safety glasses should be advised where indicated, and adequate shielding provided for machines. Proper lighting is of utmost importance throughout the plant. First-Aid stations should be established, with adequate material for handling the initial treatment of eye injuries. Lastly, it has been shown that the placement of irrigation fountains, in strategic locations in factories using chemicals, has helped materially in reducing damage as a result of chemical burns to the eye.

The instruction of parents may likewise do a great deal in decreasing the incidence of ocular trauma in children. The most common injuries are puncture wounds. The agents most often found responsible are glass, pencils, B-B shot, arrows, and sticks. All are difficult for the child to avoid and it is only through constant vigilance that youngsters can be protected.

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MANAGEMENT OF AN ACUTE HEAD INJURY

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Though much has been written on the management of the acute head injury, the subject bears frequent review and accentuation, in order to impress those responsible for the treatment of such injuries that it is the *brain* itself which must be treated. The absence of a skull fracture lulls the physician into a false sense of security and the patient is often permitted to develop a surgical hemorrhage without the benefit of intervention. It is my feeling that the neurosurgeon can more readily tell the state of the injury to the brain if given the chance to accept the responsibility. The presence or absence of a skull fracture is not an important consideration unless there is evidence of a compound skull fracture. The x-ray films can be delayed until the patient is well on the road to recovery or just before he leaves the hospital. The presence or absence of the skull fracture is not important in determining the length of stay of the patient in the hospital or the time of his ambulation. If a compound skull fracture exists, then skull films may be taken as the patient is being brought up to the operating room. It is poor surgical judgment to take the patient immediately to the x-ray department for skull x-rays unless there is indication of a compound fracture.

In the diagnosis of surgical hemorrhages within the skull, one of the most important diagnostic criteria is the change in condition of the patient from the time of the first examination. The initial examination and the initial history and findings are of the greatest diagnostic importance since they give a base line from which we can judge progression or lack of progression. The following, therefore, is the management of the patient with a head injury when brought into the emergency room. The patient is first completely undressed, so that any injuries other than craniocerebral may be recognized. If there is a bleeding wound in the scalp, a pressure dressing should be applied and the wound not sutured until the patient has been completely evaluated. The first consideration in the treatment of the patient is that of shock. Many of these patients are admitted in a state of shock, which, if due to a head injury is relatively mild and easily reversible by the usual methods for the treatment of shock. These are lowering the head, application of warmth, and the giving of intravenous fluids, plasma and/or blood.

Once shock treatment has been instituted, then an evaluation of the patient can be begun. First, it is important to note whether or not the patient was unconscious immediately after the injury; secondly, whether there was any paralysis immediately after the injury, and finally whether or not there are any injuries to the extremities, chest or abdomen. Following this perusal, the patient's general neurological condition should be evaluated. The state of the pupils, their size and reaction to light, the state of consciousness of the patient and whether or not there are any reflex changes or paralysis should be noted and written down so that the base line which is so important is recorded. If the patient is suffering from shock which is due to the head injury, then the blood pressure should rise rapidly after instituting the treatment noted above. If the blood pressure does not respond and there is persistence of severe shock, then one should look for other injuries, especially those of the chest and abdomen. When the blood pressure starts to rise, the head should be elevated and the injury to the scalp evaluated. If there are lacerations of the scalp, the area about the lacerations should be widely shaven and washed with sterile solution, then with a sterile glove the wound palpated gently to determine whether there is a compound irregular fracture in this area. The presence of a compound depressed fracture contraindicates suturing of the wound. Such a patient becomes a surgical emergency and a candidate for debridement of

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the scalp and skull. These fractures of the skull and depressions of bone often contain dirt and hair which are a source of infection. This is also true of the penetrating or perforating wounds of the head. It is mandatory then that in all of these wounds debridement must include any injury to the underlying dura or brain. If no compound fracture of the skull is found, the scalp wound can be sutured safely after the skin edges have been debrided. Buried suture material should be avoided. Subcutaneous sutures should not be used; only a through and through suture which takes in the galea.

In discussing the intracranial hemorrhages, extradural, subdural, subarachnoid, and intracerebral hemorrhages will be discussed.

The Acute and Subacute Extradural Hematoma

The acute extradural hematoma is produced by a fracture which crosses the middle meningeal artery, tears it, and allows it to bleed, or by a fracture which extends down into the foramen spinosum, thus severing the artery at that point. The bleeding which ensues is very profuse and rapid since the middle meningeal artery is a large artery. The bleeding occurs between the outer layer of the dura and the inner table of the bone. The blood gradually dissects the dura away from the inner table of the bone down into the temporal and subtemporal areas and a large hematoma forms rapidly. However, even this rapid bleeding will not produce signs of compression for at least three hours after the injury. It takes approximately three hours before a hematoma from middle meningeal artery bleeding attains sufficient size to compress the brain and produce symptoms referable to an expanding lesion. These signs are increased stupor, the development of lateralizing signs such as a contralateral hemiparesis, and a homolateral dilatation of the pupil. In addition the blood pressure rises, the pulse drops, the temperature begins to rise, and the breathing becomes stertorous and at times, irregular. The progression and development of these signs is indicative of an expanding lesion. The rapidity of the formation of these signs within three hours of the injury indicates the presence of an extradural hematoma and the patient should be immediately brought to surgery for removal of this extradural clot and ligation of the middle meningeal artery.

The subacute extradural hematoma develops in the same location, usually temporal, but is due instead to venous bleeding, either from a fracture through the diploie of the bone or from a bridging vein. This bleeding is slower than that of the middle meningeal artery and takes from six to twelve hours to develop a hematoma of sufficient size to cause symptoms. Again the signs are those of deterioration of the state of consciousness of the patient, the development of lateralizing signs, dilatation of the pupil, blood pressure, pulse, and respiratory changes. The treatment, again, is surgical evacuation of the clot and control of the venous bleeding.

Acute Subdural Hematoma

The acute subdural hematoma is difficult to differentiate from the subacute extradural hematoma. The time interval is approximately the same and a chronological development of symptoms and signs are also the same. The treatment is the same. However, in the acute subdural hematoma, one usually finds concomitant severe damage to the brain. The patient's hospital course is poor because of the brain lacerations, not because of the subdural hematoma.

Subacute Subdural Hematoma

The subacute subdural hematoma develops more slowly and gradually compresses the brain. The bleeding is less active, the time interval is usually two to six days. Since this subdural hematoma follows rather minor or moderate head injury, with or without skull fracture, and develops slowly, there is a gradual compression of the brain and there may be no clinical evidence of interference with brain function; that is, there may be no lateralizing signs, no papilledema, and no increased cerebral spinal fluid pressure. There is often some discoloration of the spinal fluid due to increased amount of protein and disin-

tegrating blood pigment. The most important clinical symptom is the poor response of the patient after two or three days of a rather mild or moderate head injury. The patient does not recover as rapidly from the injury as he should and there is a vacillation in state of consciousness. At one time, the patient may seem to be rather bright and alert; a few minutes or an hour later, he is quite stuporous or comatose; then he seems to recover, only to lapse into stupor again. This vacillation from alertness to stupor is pathognomonic of the slowly developing compressing subacute subdural hematoma. The treatment is surgical. The subacute subdural hematoma, when evacuated, leaves a markedly compressed brain which does not expand. The slow compression of the brain by the subdural accumulation of fluid displaces the brain fluid and decreases the brain volume. When the hematoma is evacuated, the brain does not expand due to the lack of the normal extra-cellular fluid. It may be necessary at times to instill normal saline solution into the spinal canal to expand such a brain since a poorly expanded brain leaves a cavity which may refill with subdural fluid. The diagnosis, therefore, of a subacute subdural hematoma is lack of normal progression of recovery, the development of lateralizing signs, fluctuations in state of consciousness, and the presence of xanthochromic spinal fluid.

The chronic subdural hematoma is very slow; it may go on to organization or even calcification. Some weeks or months after a minor injury the patient begins to develop lateralizing signs and evidence of increased intracranial pressure which, with a lack of any history of severe injury, leads to a diagnosis of possible brain tumor. On making the burrhole prior to ventriculography, the subdural hematoma is encountered and the diagnosis is made. The treatment, of course, is the same as that of acute subdural hematoma, surgical evacuation of the hematoma and expansion of the brain. It may be necessary in some cases of chronic subdural hematomas to turn a large bone flap in order to evacuate an organized hematoma.

Subarachnoid Hemorrhage

Subarachnoid hemorrhage occurs with dural laceration or a tear into the arachnoid, with or without associated extradural or subdural hematomas. There is no specific medical treatment for subarachnoid hemorrhage and certainly no surgical treatment. Subarachnoid bleeding frequently subsides spontaneously. It may lead to marked irritability of the patient and stiffness of the neck.

Patients with intracerebral hematomas secondary to brain injury usually have associated brain lacerations. These patients are severely injured and are in a serious condition when first seen. They have hemiparesis or hemiplegias and pupil changes immediately after the injury. They are deeply comatose, breathing stertorously, and show marked signs of cerebral compression. The presence of this immediate hemiparesis or hemiplegia with dilated pupil, stupor, or coma is an indication of intracerebral hemorrhage and brain laceration. There have been occasional successful surgical results in this type of hemorrhage. The evacuation of the hematoma on removal of lacerated brain tissue with control of bleeding has at times resulted in recovery of the patient. The prognosis is always grave and the likelihood of recovery is poor with such injuries.

The general management of the patient who is not a surgical problem is directed towards the cerebral edema. Therefore, supportive measures are necessary to maintain normal metabolic function. One of the most important aspects in the care of an unconscious patient is the maintenance of a clear airway to prevent asphyxia and aspiration pneumonia. The airway should be aspirated frequently. If there is evidence of obstruction, or of trachial secretions which cannot be removed by suction, then trachiotomy is indicated. Oxygen should be used as a supplement and the electrolyte balance should be maintained. Dehydration of the patient will lead to the development of acidosis and further edema and swelling of the brain. In the adult 2000 cc. of fluid daily is a minimum. The intake and output are the best criteria as to how

much fluid is necessary. It is equally important not to overhydrate the patient. In the treatment of restlessness or irritability, rectal aspirin in the unconscious patient has a twofold action. It decreases the pain and restlessness occasioned by subarachnoid blood, and in addition, is an antipyretic The dosage is from twenty to sixty grains, every six hours, depending upon the temperature and restlessness of the patient. Such patients may be restless because of a distended bladder and an indwelling catheter will effectively quiet them. In the closed head injury, the non-operative case, the use of barbiturates or morphine derivatives is contraindicated. The morphine derivatives have a tendency to increase cerebral edema and also depress the respiratory center and should, therefore, be avoided. The use of barbiturates should also be avoided because it masks the state of consciousness of the patient, one of the most important signs of the development of surgical intracranial hemorrhage. Paraldehyde may be used either rectally or intravenously. The patient should be allowed to move about, as shackles increase irritability and have a tendency to promote hypostatic pneumonia. Shackles also have a tendency to produce delirious nightmares and to increase the restlessness of the patient. Elevation of the head is important to allow for drainage downward into the spinal canal. The use of magnesium sulphate for restlessness and for edema is very effective. This medication can be given intramuscularly in two gram doses. The use of the spinal puncture should be reserved for diagnosis and not for treatment. In the immediate stages of the acute head injury, the spinal puncture is of no value, either from a diagnostic or therapeutic standpoint and may at times be quite dangerous, especially in the presence of severe cerebral edema. The spinal puncture may be used as a diagnostic procedure in the later stages of a head injury where one suspects the presence of an intracranial hematoma, and there, again, should be done with caution. If the patient remains unconscious for a longer period than two or three days, a Levine tube is inserted for feeding and medication. The vital signs of the patient must be watched very carefully for the first twelve hours, the critical period for acute of subacute extradural hematomas.

The Skull Fractures

The simple linear skull fracture does not necessitate any surgical intervention or treatment and does not prevent early ambulation of the patient; that is, if the neurological and general clinical condition of the patient warrants ambulation. The compound depressed or compound comminuted depressed fracture is a surgical emergency as described above.

Fractures through the base of the skull, associated with bleeding from the ear, nose, and mouth have no surgical implication. If the bleeding is from the ear, manipulation of the ear should be avoided, a sterile pledget of cotton should be placed in the external auditory meatus. Fractures which go through the cribiform plate and result in rhinorrhea may eventually necessitate surgery; however, the rhinorrhea often subsides spontaneously within the first 24 to 48 hours. If the rhinorrhea persists, a frontal bone flap is made and the dural laceration over the cribiform plate repaired. The presence of the rhinorrhea is a potential avenue of infection into the subarachnoid space and thus to the brain itself. All patients with rhinorrhea or bleeding from the ear must be placed upon antibiotics.

Simple depressed skull fractures are not surgical emergencies, since the danger of infection is not present. All depressed skull fractures should be elevated when the patient's condition is such that a surgical procedure will not be of danger to the patient. Elevation of the depressed skull fracture relieves compression of the underlying brain and allows for repair of laceration of the dura caused by jagged pieces of bone and the removal of bone fragments. X-ray examination of the skull does not disclose the extent or severity of the depressed skull fracture. The x-rays have a tendency to minimize the degree of depression and the surgeon is constantly amazed at the degree of destruction of dura and brain at surgery.

SUMMARY

In summary, it is suggested that early and expert evaluation of the patient be made so that one will have established a base line for future observation of the patient. Every patient should be completely examined at the time of the first examination to determine the presence or absence of complicating injuries to the viscera, extremities, and chest in addition to the cranio-cerebral injury. The shock, which is associated with head injuries, is easily combatted and the presence of irreversible shock should lead one to suspect the presence of injuries other than those confiined to the head. Shock therapy should be administered before any evaluation of the scalp lacerations are made. Laceration of the scalp should be thoroughly investigated for the possibility of compound skull fractures and if such are found, the wound should be debrided in the operating room. It is important to determine in the initial examination of the patient the presence or absence of abnormal neurological findings, pupillary changes, and the state of consciousness of the patient. The depressed skull fracture should be elevated when the patient's condition becomes good enough to allow for surgery. The treatment of the unconscious patient is that of maintenance of fluid balance, reduction of temperature, maintenance of a clear airway, and moderate sedation for restlessness. The state of unconsciousness of the patient and the progression of the state of consciousness of the patient is the paramount clinical sign in the determination of the presence or absence of expanding intracranial lesions. X-rays of the skull should be delayed unless there is a possibility of a compound fracture of the skull, and in the latter instance, x-rays are taken while transporting the patient to the operating room. Spinal puncture should be reserved for diagnosis and not for treatment.

TRAUMA TO THE ABDOMEN

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In this present age of motors, missles, and misunderstandings—both social and international—it is imperative that we as physicians constantly refresh and review the fundamental principles of traumatology; that we may be ever alert to apply them promptly, simply, logically, and efficiently.

The purpose of this article is to focus attention on "trauma to the abdomen". However, it must be emphasized that in cases of major trauma there are frequently multiple injuries. These must be initially evaluated as to their relative importance and severity. Of course, the total physiological status of the patient must constantly be kept in mind, and immediate care for the life threatening emergencies — hemorrhage, suffocation, open chest wounds, *etc.*—must be given.

The fate of the injured depends to a large extent upon the initial care that both the patient and his wounds receive. With this in mind, principles will be reviewed from the viewpoint of the surgeon first called to see the patient; that is, the surgeon on call in an emergency room.

In the management of any injury as in any disease, adequate physical examination is most important. In some instances, the character of the injury and the condition of the patient place restrictions on the comprehensive detail that is utilized in less urgent complaints; however, every reasonable attempt should be made to obtain the most accurate evaluation of the patient and his injury as soon as possible.

History as to the time of injury and type of trauma should be obtained from the patient if possible and/or witnesses when feasible. The state of consciousness determines to a very large extent the amount of accurate information the injured subject can give regarding the circumstances of his injury and his symptoms. Since the state of consciousness may change in very short periods, the examiner must take advantage of any conscious interval to gain as much information as quickly as possible.

During the physical examination the patient should be disturbed as little as possible. However, in abdominal trauma appraisal of the dorsum must not be neglected, and proper record made of all significant findings, including evidence of pertinent pre-existing diseases.

For classification purposes we may divide "abdominal trauma" into penetrating wounds, non-penetrating wounds, and thoraco-abdominal wounds (which may be either of the foregoing types.)

When the injury is of the penetrating variety, its probable nature and the structures involved are more readily surmised than when the injury has been produced by a non-penetrating force. The presence of a wound of entry and a wound of exit, if such exists, and a knowledge of the causative agent often provides sufficient evidence for a presumptive diagnosis of such injury. The intra-abdominal organs most likely to be involved depend upon the course of the missile, or upon the thrust of the penetrating instrument.

The absence of a wound of exit in the case of a bullet or the lack of factual data regarding the instrumental penetration of the abdominal cavity may hamper the above simple appraisal. Attempted probing of the wound is usually inaccurate, and misleading due to positional shifts in the various muscular layers of the abdominal wall.

Vomiting of blood strongly suggests injury to the stomach or upper intestinal tract. Blood in the urine is indicative of injury to the bladder, kidney, or ureter. Digital rectal examination disclosing blood on the examining finger directs attention to the probable injury to the rectum or lower colon. All penetrating

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wounds between the shoulders and knees should be *suspected* of abdominal cavity involvement.

Non-penetrating abdominal injuries, however, present a more acute diagnostic problem in the determination of the extent of injury to the intra-abdominal viscera. Early and accurate decisions must be made to afford either the necessary effective surgical intervention, or to preclude an unnecessary exploratory operation at a particularly inopportune time.

The X-ray evidence of air under the diaphragm is almost pathognogmonic of injury to a gas containing viscus. Auscultatory absence of peristaltic sounds is usually found if such perforation has occurred. Pain, tenderness, protective rigidity, resistance of the abdominal musculature, and a limitation or absence of abdominal respiratory excursions are characteristically present with direct injury to the parietal peritoneum or irritation due to the presence of gastrointestinal contents or blood in the peritoneal cavity.

Pain in the shoulder may be present due to the phrenic nerve distribution of diaphragmatic irritation. Most of the symptoms and signs of intra-abdominal injury associated with non-penetrating agents are of a general nature and may give little precise information. The diagnosis of non-penetrating injuries is far more difficult than that of penetrating injuries and failure to recognize visceral rupture is not uncommon. Delay or failure of diagnosis accounts in a large measure for the consistently high mortality in this type of abdominal trauma.

A simple, severe contusion may cause shock, pain, tenderness, rigidity, and most of the symptoms and signs found in the actual rupture of viscera. Ileus occurs frequently after simple abdominal contusion. It becomes obvious, therefore, that the means of distinguishing contusion from actual visceral rupture are limited.

If it is suspected that a penetrating wound involves the peritoneal cavity, it is mandatory to explore the abdomen with the least possible delay. There is a direct relationship between the time lapse from injury to operative treatment and morbidity and mortality. Occasionally it may be necessary, particularly in non-penetrating wounds, to open the abdomen without a clear idea as to the exact location of the injury; however, in either case a serious attempt should be made to locate the site of injury clinically as accurately as possible.

The most urgent circumstance for operative intervention is intra-abdominal bleeding. Indications of continued hemorrhage are: (1) Shock that does not respond to transfusion of whole blood or requires rapid, massive amounts to sustain response; (2) temporary effect from transfusion with subsequent relapse. Under these conditions immediate operation with ligation or repair of the bleeding point is required.

As a practical approach to the subject, it seems best to take up the common injuries to definite important viscera separately, recognizing that associated injuries of other structures may be present.

In thoraco-abdominal injuries, the pleural and peritoneal cavities, and the diaphragm are involved. In many instances the amount of pulmonary damage is relatively minimal and would not in itself warrant thoracotomy. However, laparotomy for injured abdominal viscera, no matter how minimal, is mandatory. In these combined injuries, priority is naturally given to correction of any discernable alteration in cardio-respiratory equilibrium before surgical repair is effected, usually through a combined thoraco-abdominal incision with repair of viscera as indicated. Individualization of cases is extremely necessary in these instances because of the varying degrees of cardio-respiratory disturbance. Difficulties at times arise in the administration of intravenous fluids because of the potential dangers of over loading a circulatory system already embarrassed by direct pulmonary trauma, hemothorax, pneumothorax, or a combination of these. Often adequate blood volume can be obtained only after an impaired cardio-respiratory imbalance has been improved or corrected.

Differential Diagnosis of Commonly Injured Viscera

The *spleen* is commonly injured by penetrating objects, such as broken ribs, and by blunt forces, as in blasts. Injuries range from minor perforations to extensive lacerations, fragmentation, maceration, and even complete detachment of the spleen from its pedicle. The diseased spleen is more susceptible to injury than the normal spleen. Because of its increased size and friability, the diseased spleen is more likely to be injured by blunt force.

Symptomatology is variable depending upon the degree of injury and the amount of intraperitoneal hemorrhage. Characteristically, one may find subjective and objective evidence of shock due to hemorrhage, as well as abdominal pain, tenderness, and rigidity—sometimes diffuse and sometimes localized in the upper left abdominal quadrant. In some instances, flank dullness or even flank fullness may be outlined. Diffuse abdominal distention, ileus, and bluish umbilical discoloration may be seen late. These findings are all indicative of gross intraperitoneal hemorrhage, rather than specific bleeding from the spleen. In many cases corroboration of the source of bleeding is made only at laparotomy; however, the possibility of injury to the spleen must be considered in all instances of trauma to the abdomen or left lower thoracic region, especially with the signs and symptoms of intra-abdominal hemorrhage. When such an injury is found, the definitive treatment is splenectomy.

The *liver* should likewise be seriously considered as an injured viscus in all considerations, particularly when the force involves the upper abdomen or lower right thorax. Severe wounds of the portal vein or hepatic vessels are rarely seen because the victim does not survive long enough to reach the emergency room. Although the actual liver wound may vary from superficial lacerations and contusions to hepatic maceration and fragmentation, the management and prognosis is chiefly dependent upon the degree of associated hemorrhage and resultant shock. In the case of missiles, careful study of the location and character of the wounds of entrance and exit, or by roentgenograms if the missile is retained, is again of importance in determining the extent of hepatic injury.

As in the case of splenic injury symptomatology is variable depending upon severity of injury. Characteristic symptoms and signs, when present, are those typical only of gross intraperitoneal hemorrhage. Icterus or bile in the urine is *not* apparent for several days. *None* of the tests for hepatic function are of any practical value in the early management of these cases. Injuries to the *extra-hepatic biliary system* are frequently associated; however, there are no distinctive signs or symptoms. Treatment again is laparotomy with repair, after anti-shock measures where indicated have been adequately employed.

Because of the great degree of mobility permitted by their mesenteric attachments, the *stomach and small intestine* are less likely to be severely damaged by blunt trauma than the solid organs. However, severe compression between the abdominal wall and the vertebral column, and blast injuries to the abdomen are known to rupture these gas and fluid filled structures.

With rupture of the stomach or small intestine, there may be hemorrhage, spreading peritoneal irritation, and shock. The characteristic signs and symptoms are therefore increased pulse rate, lowered blood pressure, nausea and/or vomiting, abdominal tenderness, and sometimes rigidity which first may be localized and later diffuse. There may be roentgenographic evidence of air under the diaphragm, as well as pain in the neck or shoulder due to sub-diaphragmatic irritation referred to these sites by the phrenic nerve. Retroperitoneal or mediastinal emphysema may be noted when the retroperitoneal portion of the duodenum has been injured. Blood in the vomitus or aspirated gastric fluid is suggestive of upper intestinal injury.

Penetrating injuries by various missiles and weapons are not only prone to involve the stomach and small intestine, but frequently are guilty of multiple per-

forating visceral wounds; numerous intestinal coils may lie in the route of the injuring agent. Location of wounds of entrance and exit, when present, may help to pre-determine the viscera involved. However, bullets deflected by bony structures may assume amazing routes: wounds of entrance or exit may be remote from the abdomen, even in the extremities, thorax, or neck.

The associate signs and symptoms are again those due to hemorrhage, gross peritoneal contamination, and shock. Massive hemorrhage may be precipitated by injury to the mesenteric vessels. Tears in the mesentary may be extensive enough to deprive segments of the intestine of its blood supply. These injuries likewise demand immediate laparotomy and thorough inspection as soon as it is feasible, with repair of whatever intra-abdominal damage that may be found.

Injuries to the *colon and rectum* are usually indistinguishable clinically from similar injuries to other parts of the gastro-intestinal tract. Diagnostic efforts, therefore, are directed more toward determining whether a perforation exists, rather than establishing its location.

Pancreatic injury occurs most commonly in association with injury to other viscera. This is because of the excellent protection afforded by its position within the abdomen. Diagnosis of isolated pancreatic injury is not commonly warranted, but its consideration should accompany all instances of trauma to the upper abdomen. Injury to the pancreas usually consists of incomplete or complete rupture of the organ, ranging from sub-capsular edema and hematoma to gross hemorrhage, loss of pancreatic fluid, and peritonitis. Pancreatic pseudocysts of enormous proportions have followed. The blood serum amylase may be elevated considerably due to traumatic pancreatitis. Elevation of the diaphragm or displacement of the stomach can result from hemorrhage or accumulation of fluids. Exploratory laparotomy for repair of other viscera should always be accompanied by inspection of the pancreas.

Injuries to the *kidney* and *bladder* should be included in any review of trauma to the abdomen in spite of their extra-peritoneal locations. Rupture of the bladder, particularly when filled, can occur easily with blunt trauma. In all cases of fractured pelvis, especially those involving the pubic arch or rami, immediate examination for bladder damage should be carried out. Likewise in all penerating injuries of the lower abdomen the bladder should be investigated.

Extra-peritoneal rupture of the bladder causes extravasation of urine into the loose areolar tissue of the space of Retzius with severe phlegmonous destruction of the infiltrated tissues and extreme general toxemia from local absorption. When the fundus of the bladder is ruptured, the leakage is intraperitoneal, and consequently a boardlike rigidity will develop from peritoneal irritation.

In all cases where bladder injury is suspected, the bladder should be immediately catheterized with sterile precautions and the amount and character of fluid scrutinized. If clear urine is obtained, the bladder is not damaged. If a sizeable amount of urine is obtained. even though it is bloody, there is reasonable assurance that nothing more than bladder contusion has occurred. Usually the urine clears shortly with indwelling catheterization for a few days.

If the catheter enters the bladder readily, and nothing is obtained but a little watery or clotted blood, bladder rupture is almost a certainty. Confirmation can be obtained safely by instilling a small measured amount (100 to 200 cc.) of sterile water through the catheter and comparing the amount immediately retrievable. If little or none can be recovered, bladder rupture is indicated.

Extravasation is stopped by immediate cystotomy. Associate drainage of the areolar tissue is necessary. The wound of the bladder is of third importance and may be simply sutured.

Diagnosis of injury to the kidney is usually made by the presence of hematuria combined with history of injury and pain in the flank. In the majority of instances the bleeding subsides under

conservative management. Persistent progressive hemorrhage or obvious maceration of the kidney occasionally requires nephrectomy.

SUMMARY

A brief review of the fundamental considerations of trauma to the abdomen has been presented. Emphasis is placed on the fact that the fate of the injured depends to a large extent upon the initial care that both the patient and his wounds receive. Promptness, simplicity, logic, and efficiency on the part of the surgeon who first sees the patient is emphasized. He must consider the following:

1. Prompt appraisal of general condition of patient.
2. Immediate care for any life threatening emergencies that exist, *i.e.* hemorrhage, suffocation, open chest wounds, *etc.*
3. History of injury.
4. COMPLETE DIAGNOSTIC PHYSICAL EXAMINATION with the determination of the likelihood of a visceral injury.
5. The individualization of cases and a constant regard for the total physiologic status of the patient.

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MANAGEMENT OF ACUTE DISLOCATIONS OF THE EXTREMITIES

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The liability of any individual joint to suffer a dislocation depends upon its exposure to injury, the shape of the articular surface, and the degree of support given to it by supporting ligaments and muscles. We find, therefore, that the shoulder joint is commonly dislocated because the glenoid cavity is shallow, and the support given by the surrounding muscles and ligaments is comparatively lax. The hip joint, on the other hand, is less commonly dislocated because the cup-shaped acetabulum is deep, and the muscles closely support the joint.

Clinical Features

I shall begin by giving only a few of the general clinical features relating to the diagnosis of a dislocation, leaving the more detailed signs and symptoms to the discussion of the individual dislocation which follows.

Pain is due to local trauma or pressure on nerves, as for example, the head of the humerus impinging on the brachial plexus.

Loss of Function. Fixity replaces mobility.

Deformity. The limb may be shortened or lengthened, or the presence of abnormal alignment may be evident.

Abnormal Position. The end of the bone can be detected in an abnormal position. This is the *absolute* sign of a dislocation. Unless the dislocation is accompanied by a fracture, movement of the shaft of the bone causes corresponding movement of the articular end.

In the following pages I shall attempt to discuss the management of the dislocations of the upper and lower extremities that may confront the interne on a fracture ward. Wherever possible, I shall also include some of the difficulties which I personally experienced while endeavoring to reduce a dislocation, as well as some of the sins of ommission.

Acute Dislocations of the Upper Extremity

The most common dislocation of the upper extremity is the dislocated shoulder. This type of injury in an individual under twenty years of age is extremely rare. History is of extreme importance in the diagnosis of this injury. The patient, if able, would give a history of falling on an outstretched hand, followed by pain and the inability to move the shoulder of the involved side.

On examination, the affected shoulder would not have its normal rotund outline and the acromion process would be unduly prominent. This would be accompanied by difficulty in putting the elbow to the involved side. These are the major clinical signs that are to be found in a sub-coracoid dislocation, which is the most common position for a forward dislocation of the humeral head.

Pathology

The head of the humerus is driven forward and in the majority of cases the capsule is avulsed from its attachment to the glenoid cartilage. Sometimes the capsule may be avulsed from its humeral attachment. The head of the humerus then comes to lie below the glenoid, or in front of the glenoid beneath the coracoid process. On very rare occasions, the head may be displaced backwards by a direct blow over the front of the joint or by a violent internal rotation movement. The backward dislocation will cause the capsule and labrium to be torn from the posterior margin of the glenoid and the humeral head will come to lie behind the glenoid in a subspinous position.

Treatment

Before one institutes any definitive form of treatment, it is advisable to exclude any concomitant neuro-vascular

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injury. Not uncommonly, the circumflex nerve which innervates the deltoid muscle may be injured. This may be ruled out by requesting the patient to abduct the shoulder while the examiner palpates the deltoid muscle for the presence of contraction. The axillary vessels may be pressed upon by the head of the humerus, causing the hand to become cold and blue until reduction is obtained.

There are various methods of reducing a subcoracoid dislocation, but I must first stress that before any procedure is attempted the patient must be thoroughly relaxed under a general anesthetic. Although morphine or demerol may at times give sufficient relaxation for a successful reduction, it is my experience that the procedure is easier for both the patient and the physician if the reduction is attempted under general anesthesia. I have also used brachial block anesthesia with some success, but this too is not without its dangers because of the proximity of the subclavian artery which may be pierced in an attempt to reach the brachial plexus.

As far as the method of reduction, I would advise that one should learn one procedure and stick to it. As far as I am concerned, the Kocher manipulation has worked well for me, and I shall therefore describe it in detail.

The head of the humerus is held in an inwardly displaced and an inwardly rotated position by the tension of the subscapularis muscle. To reduce the dislocation, therefore, this muscle must be stretched slowly and gradually. For a dislocation of the right shoulder, the surgeon takes the elbow in his right hand and the wrist in his left hand. Smooth and firm traction is applied to the humerus by pulling with the right hand. The arm is then very slowly and gently externally rotated by moving the wrist outwards until the normal limit of ninety degrees of external rotation is reached. Keeping the limb in full external rotation, the limb is adducted across the chest. Finally the limb is internally rotated and the hand is brought over to the opposite shoulder.

After reduction it is most important that the shoulder be immobilized in a Velpeau cast for a period of three weeks to prevent re-dislocation and to permit adequate healing of the soft tissue structures around the shoulder joint.

If the Kocher method fails, more power may be obtained by the Hippocratic method. The surgeon grasps the wrist with both hands and places his stockinged foot between the upper arm and chest. With the surgeon's knees and elbows straight, he leans backwards, thus applying traction. He slowly externally rotates the limb and then adducts it over the leverage provided by his foot. Again, once reduction has been accomplished, the shoulder must be immobilized for a period of three weeks.

Dislocation of the Elbow

Blount states that dislocation of the elbow without fracture is more common in children and forms about six per cent of all elbow injuries in children. However, these injuries do occur in adults, usually of both the radius and ulna, which dislocate to a posterior position. Sometimes the injury is associated with avulsion of the internal epicondyle or its epiphysis. This is important to recognize before attempting reduction in order to prevent the fragment from being imprisoned in the joint.

Anterior dislocations of the elbow are less frequent and if it occurs, it is the radius that dislocates forward with an associated fracture of the ulna (Monteggia fracture).

Treatment

General anesthesia is advisable for treating a posterior dislocation of the elbow. Traction is applied to the forearm and the elbow is gradually flexed. The radial pulse should be checked after reduction. Hyperflexion of the elbow may embarrass radial circulation. The elbow should be immobilized in a position of optimum flexion with a cast extending from the wrist to below the axilla for a period of three weeks. If immobilization is not carried out at the time of reduction, one may be extremely embarrassed to find that the elbow has re-dislocated as the patient is being returned to the ward.

Dislocation of the Lunate Bone

The semilunar (lunate) bone is dislocated forward beneath the anterior

annular ligament. This causes the flexor tendons to be pushed forward and, therefore, the patient is unable to move the semi-flexed fingers. In about fifty per cent of patients there is also a median nerve paralysis. Examination of the wrist may only reveal it to be swollen and painful without any obvious deformity.

The X-ray appearance of this dislocation is typical. On lateral view of the involved wrist, the head of the capitate bone no longer lies in the cup of the semilunar bone but is displaced behind it. In the anterior-posterior view, the semilunar bone has the appearance of being triangular instead of its normal quadrilateral appearance.

Treatment

Reduction is usually simple. Once again the patient should receive a general anesthetic. The surgeon presses the front of the semilunar bone with the thumb, while, with the other hand grasping the patient's fingers, the physician applies strong traction to the remainder of the wrist bones, pulling the capitate away from the radius and gradually flexing the wrist, so that the head of the capitate is pulled into the cup of the semilunar bone. The wrist is then immobilized in a cast in a forty-five degree palmar flexion for about ten days to two weeks.

Dislocation of the Lunate and Half of the Scaphoid

Sometimes associated with a dislocation of the semilunar bone, there may be an associated fracture of the waist of the scaphoid, and the proximal half of the scaphoid may be dislocated with the semilunar bone. The distal half of the scaphoid remains attached to the capitate and other carpal bones. The reduction is carried out in the same manner as for a dislocated semilunar bone, but immobilization in a plaster cast is carried out for several months.

Dislocation of the Fingers

Dislocations are caused usually by hyperextension and a small triangular chip may be broken off from the base of the distal phalanx. A digital block is usually sufficient and reduction is carried out by traction and flexion. The finger is immobilized in a position of forty degree flexion for a period of three weeks. (If the attached fragment is large, traction should first be carried out.)

Dislocation of the Thumb

Dislocations of the metacarpo-phalangeal joint of the thumb are also produced by hyper-extension. They are sometimes difficult to reduce, because the tendons of the short thenar muscles slip around the sides of the metacarpal head. If manipulative reduction fails, then surgical intervention is necessary.

Dislocation of the Hip

The hip may be dislocated posteriorly or anteriorly. The most common cause of a posterior dislocation is the "dashboard" injury. The patient usually sits next to the driver with the hips flexed and the thigh adducted. Very often one knee is crossed over the other, and therefore, the knee nearest to the dashboard in a collision will hit the dashboard, thus driving the femoral head posteriorly behind the acetabulum. When the patient is seen, the limb is in adduction and internal rotation.

An anterior dislocation occurs usually when the hip joint is widely adducted as in falling from any height. In this case, the femoral head is thrust forward in front of the acetabulum. When this latter patient is seen, the involved limb will be in external rotation. An anterior dislocation is sometimes difficult to distinguish clinically from a fracture of the hip: roentgenograms must be taken to differentiate the two entities.

Methods of Reduction

The patient must be thoroughly anesthetized, lying on blankets placed on the floor. The hip is then flexed, turned into a neutral position, and the head of the femur lifted into the acetabulum. This is a simple method and may be used for both anterior and posterior dislocations. Following reduction, the patient must be placed in a hip spica for a period of six to eight weeks and then watched carefully for the possibility of an aseptic necrosis of the head of the femur.

Dislocation of the Knee

Dislocation of the knee is usually caused by direct violence to the head of

the tibia or by a twisting or hyper-extension strain. The knee may be dislocated posteriorly, medially, or laterally. There is an associated tear of the cruciate and collateral ligaments and a meniscus injury. Treatment under anesthesia is usually simple. Reduction is accomplished by traction of the leg.

The limb is then immobilized in a plastercast for a period of three months. During this time, quadriceps exercises should be encouraged and weight bearing with the cast on may be allowed after six weeks. If the strength of the quadriceps is good, the patient may be saved further surgical intervention because of instability of the knee joint.

Dislocation of the Ankle Joint

Dislocation of the ankle joint is usually associated with fractures around the ankle and as this is the more common occurence than just a pure dislocation without any fracture, I shall discuss the fracture-dislocation first.

Outward Displacement

Outward displacement of the astragalus (tabus) occurs in injuries of the ankle where the foot is forcefully externally rotated or abducted in relation to the leg. The severity of the force determines whether the external malleolus will be fractured and displaced laterally, or whether both malleoli will be fractured together with outer displacement of the astragalus and foot.

If the force is very severe, the astragalus may be displaced posteriorly, causing at the same time a fracture of the posterior margin of the tibia. This is then known as a trimalleolar fracture. The dislocation of the astragalus will then be not only lateral but also posterior and this must be taken into consideration in the treatment.

Treatment of Outward Dislocation with Fracture of the External Malleolus and/or Bimalleolar Fracture

With the patient anesthetized, and the limb hanging over the end of a table, a cast is applied from the toes to just below the knee. While the plaster is hardening, the surgeon supports the forefoot with his knee to keep it at right angle with the leg and then uses his hands to push the astragalus inwards as far as possible towards the internal malleolus.

For example, in the case of a fracture dislocation of the left ankle, the left hand is placed over the lower shin, and the right hand over the outer side of the ankle and heel. Firm, steady pressure is applied over the malleolus. It is usually impossible to over-reduce a bimalleolar fracture unless the fractured medial malleolus is an unusually large fragment. The ankle is usually immobilized for a minimum period of ten weeks.

Treatment of a Trimalleolar Fracture with Outward and Backward Dislocation

The anesthetized patient is positioned as for a bimalleolar fracture dislocation. For example, in a trimalleolar fracture dislocation of the right ankle, the surgeon places his right hand over the front inner aspect of the lower shin, and the left hand over the lateral aspect of the ankle with his fingers curved around the back of the heel. The foot is pulled strongly forwards to overcome the posterior dislocation and pressed strongly inwards to overcome the outward dislocation. This must all be done before the plaster has had a chance to harden.

The accuracy of the reduction must be confirmed with post-reductive roentgenograms. If the reduction is unsuccessful, another attempt must be made after removing the cast. In a trimalleolar fracture, the cast should extend above the knee.

Inward Displacement

Inward displacement of the astragalus with fractures of the malleoli are due to a forcible inversion of the foot which drives the astragalus against the internal malleolus. The severity of the inward force will determine whether one or both malleoli are fractured with inward displacement of the medial malleolus, astragalus, and foot. If the force is very severe, the posterior margin of the lower end of the tibia will be fractured, thus giving both a posterior and inward dislocation of the foot.

Treatment for Inward (adduction) Fracture Dislocation

For this injury the principle of treatment is exactly the same as in outward

(abduction) fracture-dislocation. The astragalus is replaced by strong outward pressure applied over the internal malleolus and inner side of the heel. If there is a posterior dislocation present as well, as in a trimalleolar fracture dislocation, this must be reduced in a similar manner as described above; that is, by passing the fingers around the heel of the foot, and pushing forward.

A cast extending from the toes to above the knee is applied with the foot in an inverted position. The cast remains on for a period of at least ten weeks. Roentgenograms should be taken monthly to follow the healing process.

Forward Dislocation of the Ankle Joint

This type of dislocation is usually associated with a fracture of the anterior margin of the tibia. It is, as a rule, due to a fall from a height, so that the foot is driven forwards and upwards. Sometimes the lower end of the tibia and both malleoli are extensively comminuted.

Treatment

Usually a large fragment of bone is broken off the anterior margin of the tibia at its lower end, and this fragment, together with the astragalus and the other tarsal bones, is displaced forwards and upwards. This fracture dislocation, therefore, must be reduced by pushing the tarsus backwards and downwards and immobilizing in a plaster cast with the foot in moderate plantar flexion for a period of not less than ten weeks.

If reduction is unsuccessful, open reduction with fixation of the broken anterior fragment with a screw must be performed.

Dislocation of the Subtalar Joint

This type of dislocation is caused by a severe inversion strain with the foot plantar flexed. The tarsal bones are dislocated inwards while the talus remains undisturbed between the medial and lateral malleoli. This occurs if only the interosseous ligaments of the subtalar joints are torn. If, however, all the ligaments are torn, including the lateral ligaments of the ankle, there will also be a total dislocation of the talus. The dislocation of the talus may rupture through the soft tissues.

Treatment

Reduction of a total dislocation of the talus may be more difficult. The foot of the anesthetized patient should first be inverted and plantar flexed and then the surgeon, with the foot in this position, should press with both thumbs on the posterior part of the talus, pushing the talus inwards and backwards, at the same time trying to correct its rotation around the axis. If this fails, a pin may be placed through the os calcis and traction applied.

The foot should be immobilized in right angled dorsi-flexion, and weight bearing should be delayed for several months to prevent aseptic necrosis of the talus with a resulting traumatic arthritis.

SPECIAL ARTICLES:

CURRENT VIEWS REGARDING COLLAGEN DISEASES AND PREGNANCY

S. J. TURNER, M.D.*

Diffuse collagen[1,2,3,4,5,6] diseases are a group of systemic maladies of unknown causation which are characterized by similar histopathologic lesions consisting of mucoid or fibrinoid degeneration of the collagenous portion of connective tissue. Each one of the diseases has general widespread involvement of the connective tissue with a predilection for specific organs. The clinical manifestations depend upon the extent of the pathologic processes and upon the organs involved by the disease. Since connective tissue is universal it can be readily understood why such common manifestations as arthritis, arthralgia, erythematous or purpuric skin eruptions and symptoms referrable to various viscera, particularly cardiac and renal in character, are frequently present in most of these diseases. Another common and extremely important feature is the favorable response observed when steroids are administered to those afflicted with a collagen disorder.

It was Klemperer and his associates[5] who first coined the term "collagen disease" when they observed fibrinoid degeneration in the collagen found in the intercellular spaces of connective tissue as a primary feature of the histopathologic characteristic of most of these disorders. These authors included disseminated lupus erythematosus, diffuse scleroderma, and dermatomyositis into the group of collagen diseases. Periarteritis nodosa, acute rheumatic fever, rheumatoid arthritis, and serum sickness were later added to this group of diseases. This new concept of a malady which involves primarily not the cellular elements but the mesenchymal tissue was completely new and gave rise to additional interest and study into the nature of the physical and chemical properties of the substances which occupy the intercellular spaces.

The normal components of connective tissue consist of cells, ground substance, fibers, and basement membrane. The cells are mostly fibroblasts and macrophages whose function may be to aid in the production of ground substance. The ground substance is an amorphous, plastic material which assumes the form of a gel. This substance lies between the cells and the fibers and binds the minute fibrils into larger fibers which become histologically visible; its chemical products are mainly mucopolysaccharides, notably hyaluronic acid and chondroitin sulfuric acid. Hyaluronic acid is most abundant in synovial fluid, vitreous humor, and umbilical cord. It binds water and is responsible for the hydrophillic character of ground substance. Chondroitin sulfates are more abundant in the firmer types of connective tissue such as cornea, cartilage, tendons, bones, and tissues in heart valves and fascia. Collagen proteins are found mostly in the cement substance rather than in the fibrils or fibers of the intercellular spaces. The fibers are collagen, reticular, and elastic in nature. Of these three materials, more is known about collagenous fibers because of their greater abundance; they make up approximately one-third of body proteins, including the greater part of cartilage, tendon, and bone. Elastic fibers are found chiefly in the walls of blood vessels and in the ligamentum flavum. Reticular fibers form a delicate network which support mainly the parenchyma of organs, such as spleen or liver. Chemically, collagen is a relatively insoluble protein and consists mainly of three amino acids:

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glycine, proline, and hydroxyproline. The normal physiologic function of the various components of connective tissue is not definitely known. It is believed that the ground substance acts as a medium for the transmission of metabolites to and from the plasma and connective tissue cells. Ascorbic acid and various other vitamins are essential for the formation of the collagens and mucopolysaccharides of connective tissue. The growth hormones of the anterior pituitary is essential for the normal development of connective tissue, whereas, high doses of cortisone suppress its formation.

The morphologic change which may occur within the intercellular spaces of connective tissue following any kind of injury may consist of degeneration and necrosis, multiplication of fibers and sclerosis, and proliferation of cells. Mucoid degeneration is an early manifestation of all collagen disorders and it is the result of the accumulation of acid mucopolysaccharides, mainly hyaluronidase, within the framework of connective tissue. It is most likely the result of increased fibroblastic activity. Fibrinoid denegeration and necrosis occur as the disease progresses and are characterized by deposition in the intercellular spaces of a homogeneous, eosinophillic, highly refractile bandlike material which has the staining properties of fibrin. The exact components of fibrinoid are not known but it is believed that most likely it is formed from either one of the mucopolysaccharides and alkaline proteins derived from blood plasma, *i.e.*, fibrinogen, serum protein, or from injured connective tissue elements; however, it may be that it is derived from both sources. Fibrinoid is a precipitate formed in the ground substance and in the cement portion of basement membrane and collagen fibers. The intensity and extent of the morphologic changes of the connective tissue varies with the type and extent of disease. Some organs may have extensive necrosis of their connective tissue elements which is usually followed by infiltration with leukocytes, plasma cells, lymphocytes and by proliferation of fibroblasts leading ultimately to sclerosis.

There are also various changes in the blood and urine which may be commonly observed in most collagen diseases. Anemia is a predominating feature; the sedimentation rate is usually elevated. Liver damage should be suspected whenever the serum albumin and the collagenase inhibitor are depressed. Due to mesenchymal damage there is an elevation of serum fibrinogen, serum alpha globulin, serum mucopolysaccharides, and serum hyaluronidase inhibitor, (perhaps heparin). Serum gamma globulin is elevated in all collagen disorders with the exception of serum sickness. When the kidneys are affected a telescopic urine may be present.

The recognition of a specific collagen disorder may be somewhat difficult during the early phase of the disease. However, being aware of the general as well as the specific characteristics of collagen disorders will no doubt aid in the establishment of a correct diagnosis. Since it is not within the scope of this presentation to enumerate in detail all there is to know about collagen disease, it is felt that perhaps by just mentioning the prominent features of the dyscollagenosis which have been known to occur with pregnancy may be of value.

Acute rheumatic fever usually follows an acute upper respiratory infection caused by a hemolytic streptococcus and occurs mostly in children between eight and fifteen years of age. The onset is rapid with fever, swollen and painful joints. Cardiac manifestations may follow. Autopsy findings show the characteristic Aschoff bodies in the myocardium.

Rheumatoid arthritis occurs most commonly in women between twenty and forty years of age. The onset is gradual though it may be acute. The joints and adjacent structures are primarily affected. The heart, kidneys, nerves, and skin may also become involved. In the joints there is an overgrowth of the synovial endothelium and the underlying stroma with the infiltration of plasma cells, leukocytes, lymphocytes, and eosinophils. Subcutaneous nodules are frequently present and are characterized by a central area of fibrinoid de-

generation and necrosis surrounded by a zone of pallisading spindle cells, probably fibroblasts surrounded by connective tissue containing lymphocytes and other elements. The perineal nerves and the glomerular tufts are occasionally involved.

Disseminated lupus erythematosus[17] is a serious systemic disease of unknown etiology. It occurs most frequently in women between the second and fourth decades. It usually ends fatally in from one to five years. Most endothelial and serous surfaces are affected. The lesions are in the walls of the smaller arterioles of the skin, joints, myocardium, endocarium (Verrucous Endocarditis of Libman-Sacks), and kidneys (wire looping of basement membrane of the glomeruli). The intra-abdominal viscera, the lymph nodes, as well as the blood forming organs are frequently affected. The presence of lupus erythematosus rosettes and cells provide reasonable certainty in the diagnosis of this disease. L-E cells may be found in the peripheral blood, bone marrow, and frequently within the parenchymatous organs at autopsy. These cells are leukocytes containing injested nuclear material from desoxyribonucleic acid. It is believed that the L-E cells are produced by a specific protein with antigenic properties which is to be found in the gamma globulin fraction of patients affected with disseminated lupus erythematosus; this specific protein is known as the Haserick factor. The clinical manifestations are characterized by general malaise, septic temperature, weight loss, weakness, arthritis, arthralgia, Raynaud's phenomenon, and edema. Generalized adenopathy, hepatomegaly, splenomegaly and various cardiac, renal and cerebral manifestations occur frequently as the disease becomes more severe. Evidence of suppression of the hematopoietic system is invariably present. The course is variable with remissions and exacerbations. Without steroid therapy only twenty to twenty-five per cent of patients remain alive for a period of five or more years.

Generalized scleroderma, dermatomyositis, serum sickness and periarteritis nodosa will not be dealt with in detail because no references could be found in the literature regarding their relationship to pregnancy. Recent publications have made some reference to the effect of pregnancy on acute rheumatic fever, rheumatoid arthritis, and disseminated lupus erythematosus.

Holman and Jones[7], experimenting with dogs, found that with certain dietary measures they could with regularity produce arterial lesions resembling those of periarteritis nodosa and rheumatoid arthritis. Under similar circumstances these lesions could not be produced in pregnant animals. They, therefore, concluded that pregnancy has a protective influence against collagen disorders. Recently Oka[9], from Finland, observed 732 women with rheumatoid arthritis during pregnancy or early in the post partum period. He found that symptoms may subside during pregnancy, that they become worse soon after the termination of pregnancy, and that most frequently they reappear in a much more aggravated state than prior to gestation. Some cases of rheumatoid arthritis occurred during pregnancy or soon after delivery. Oka, therefore, concluded that enough cases appeared so closely in relation to pregnancy that in certain circumstances one can almost regard pregnancy as an etiologic factor. Gould[8] reported three cases of rheumatoid arthritis, two of which were exacerbated and one of which had its onset during pregnancy. All of these cases responded well to small doses of cortisone without any ill effect on the pregnancy.

In view of the fact that most cases of acute rheumatic fever occur before the childbearing age, comparatively few reports are available for study. Hollander and Goldsmith[10] report a case of acute rheumatic fever which occurred in a twenty-nine year old, Para O, Gravida I fifteen days before the expected date of delivery. This patient improved under treatment with salicylates but did not make a full recovery until after she was delivered. Hollander and Goldsmith also quote a similar experience of Hefferman and Schaeffer. In Hefferman's case the pregnancy had to be terminated before the patient could respond to therapy.

Schaeffer's patient delivered four days after the onset of acute rheumatic fever; the infant developed polyarticular arthritis a few days after delivery. Singh and Miller[12] recently reported a case of acute rheumatic fever which occurred during the thirty-sixth week of gestation in a Para III, Gravida V. This patient was successfully treated with ACTH, 100 mgms. or less per day and made a complete recovery after delivering an apparently healthy infant. Sudden death from acute rheumatic carditis was reported in one case during labor by Kennedy[11] who also mentioned two similar cases reported by McKeown that died soon after delivery. The symptoms in all of these cases were not remarkable, but autopsy studies revealed definite evidence of acute rheumatic myocarditis with marked involvement of the coronary arteries which most likely was responsible for the sudden death.

In spite of the fact that ninety-five per cent of disseminated lupus erythematosus occurs in women during the childbearing age, only a few case reports of pregnancy associated with systemic lupus erythematosus have thus far appeared in the world literature. Crawford and Leeper[8] report three cases of local discoid lupus erythematosus which were complicated by pregnancy. All of their patients had an exacerbation of the disease during pregnancy. One patient had an aggravation of the dermatologic lesions which improved one month post partum. In another case, the disease became acutely disseminated and the patient died one day after delivery of an infant which remained alive. The disease in the third patient also became systemic and a stillborn fetus was delivered during the eighth month of gestation; this patient died five years later from an acute exacerbation of systemic lupus erythematosus. These authors came to the conclusion that pregnancy is not a good omen for women afflicted with lupus erythematosus.

Ellis and Bereston[13] had the experience of treating three pregnant women who were victims of systemic lupus erythematosus. All of these women and their infants failed to survive. These authors reviewed the literature and also summarized the reports of 100 cases they received by sending out a questionnaire to 280 dermatologists. From their study, Ellis and Bereston concluded that pregnancy has no adverse effect on chronic discoid or systemic lupus and that the prognosis depends upon the stage of the disease. Donaldson[15] refers to fifteen cases he found in the literature and reports five additional cases of systemic lupus erythematosus which he observed during pregnancy. This author states that "patients with lupus erythematosus tolerate pregnancy without adverse effect on the disease." Merrill[18] presents a case of disseminated lupus erythematosus who conceived while receiving 100 to 150 mgms. of cortisone per day. The pregnancy was not suspected and not recognized until the twenty-fourth to twenty-sixth week. The patient's condition grew progressively worse in spite of the fact that high doses of hydrocortisone were administered at all times. The patient died six days after an abdominal hysterotomy at six and one-half months' gestation. The infant weighed 980 grams and survived for approximately twenty-four hours. Except for prematurity, the infant had no other abnormalities. Arje and Bachman[19] report a case of acute disseminated lupus erythematosus with two pregnancies and a favorable outcome for the mother and infants. The second pregnancy occurred while the patient was receiving between twenty-five to fifty mgms. of cortisone per day.

Recently in collaboration with three co-authors[16, 17], I had the occasion to review the literature and to add two additional cases of disseminated lupus erythematosus during pregnancy. After evaluating our own experience, as well as the available data, we came to the conclusion that any form of lupus erythematosus may tolerate pregnancy well provided the disease is under control and in a quiescent state. Generally, it was our feeling that any exacerbation or change from a chronic discoid or from a quiescent state to that of the systemic disseminated type of lupus erythematosus which cannot be controlled medically within a reasonable period of time

may require an interruption of pregnancy. Somehow we failed to recognize the true nature of the disease in our fatal case until the patient was pre-terminal. One cannot help being impressed by the fact that the pregnancy aggravated the course of the disseminated lupus erythematosus. Our second case was known to be afflicted with systemic lupus erythematosus; she was in a quiescent state and under continuous treatment with cortisone since April, 1951. This woman has given birth to three normal living infants during the past three years and is getting along quite well on a maintenance dose of fifteen to twenty mgms. of cortisone per day.

COMMENT

Originally Hench[8] (quoted by Gould) introduced steroids as a therapeutic agent in rheumatoid arthritis because he assumed that pregnancy suppressed the progress of the disease. Nevertheless, more recent reports have shown that rheumatoid arthritis may be aggravated or even have its onset during pregnancy. The rare occurrence of acute rheumatic fever during pregnancy may perhaps be best explained by the fact that disease occurs most frequently before the childbearing age. The cases of rheumatoid arthritis and acute rheumatic fever which have been reported in association with pregnancy would seem to dispel the idea that pregnancy offers an *immunity* against these diseases. Furthermore it has been shown[8,9] that treatment with salicylates and with steroids aids in the control of the disease and that complete recovery from these collagen diseases usually occurs after the pregnancy is terminated. As for disseminated lupus erythematosus, little is known about the exact relationship between pregnancy and the course of the disease. There are those who believe that the disease is not affected by pregnancy. However, careful analysis of the reported cases reveals that pregnancy is not desirable in women afflicted with uncontrolled systemic lupus erythematosus. The summary provided by Ellis and Bereston[13] indicates a maternal mortality of twenty-five per cent and a fetal mortality of thirty per cent in acute cases; in subacute cases there was no maternal mortality but the fetal mortality was forty-six per cent. A summary of the cases which were reported in detail[17] reveals that in the acute systemic disease eight out of ten patients became critically ill during pregnancy; five patients died early in the post partum period; one patient remained chronically ill and died six years later. Only one infant survived among these six patients. Among the more recent reports[14] there is enough evidence that cortisone or other steroid therapy to the mother does not affect the infant. There is also an indication that most of these patients[15] will continue to get along fairly well while taking cortisone and/or ACTH. Occasionally an acute exacerbation of the disease may occur in spite of steroid therapy. The evidence, even though still limited in scope, points to support the original belief of Crawford and Leeper[14] who stated that pregnancy is not a good omen for women with extensive lupus erythematosus. I, therefore, am of the belief that pregnancy may continue in the presence of systemic disease but only when the disease is in a state of remission and under complete medical control. Any exacerbation of the disease which cannot be controlled should be enough indication to consider interruption of the state of gestation and should be undertaken early enough to prevent irreversible damage to the kidneys, vascular system, and other vital organs of the body. One should always be on the alert for the possibility of a superimposed toxemia which will most likely be less responsive to therapy in the presence of disseminated lupus erythematosus.

SUMMARY AND CONCLUSION

1. The nature of collagen diseases and the present day views regarding their relationship to pregnancy have been given.

2. Rheumatoid arthritis and acute rheumatic fever may have their onset or become aggravated during pregnancy. These diseases respond to salicylate or even more so to steroid therapy without any ill effect upon the pregnancy.

3. The available data discloses that before the advent of steroid therapy,

pregnancy was not a good omen for women who were afflicted with systemic lupus erythematosus.

4. The introduction of steroid therapy in acute and subacute disseminated lupus erythematosus seems to be of value in controlling the disease. More women may therefore live long enough to become pregnant while having the disease.

5. Even though pregnancy may be well tolerated when the disease is under control medically or in a state of remission, nevertheless, great care is to be exercised in observing these patients.

6. It is strongly suggested that termination of pregnancy may have to be considered whenever the disease becomes aggravated and cannot be controlled with medical management.

7. A superimposed toxemia should be treated actively at all times because disseminated lupus erythematosus becomes worse in the presence of toxemia of pregnancy.

8. Pregnancy may occur while women are under steroid therapy.

9. In cases of pregnancy when the disease is well controlled with steroid therapy the infants appear to be normal.

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CHEMICAL TESTS FOR INTOXICATION*

DANIEL T. DRAGEL**

Human consumption of alcohol in its varied forms since the repeal of the Volstead Act has increased sharply from year to year. Why the public responded with enthusiasm and favor to a costly social habit, detrimental to health, and at times detrimental to the well-being of society, is difficult to determine. It may be that the unique prohibition phrase "for medicinal use only" restrained the public for a temporary period until the emergence of a new era wherein the medical profession cast aside the privilege of writing prescriptions for whisky, *capiat ut dictum* and the counter salesman stated that we have a special on "Calverts, the breakfast of champions."

In this new era, driving habits of the public were profoundly influenced by a change in horsepower, and in the fact that the driving public was imbibing quantities of alcohol in order to bolster enough courage to take to the road.

Law enforcement procedures in vogue for many years could no longer be adapted to changing conditions. Accident reports indicated that the wholesale slaughter of humans on the highways was caused by reckless driving, driving on the wrong side of the road, speeding, and improper turning. Other sources of information tended to suggest that the maiming of our citizenry on the highways was caused by a lack of courtesy and understanding amongst drivers and pedestrians. Of the various direct causes of accidents, it cannot be denied that alcoholic influence is a potent factor in sparking the chain of events preceding these accidents.

In endeavoring to apply a yardstick as a measure of alcoholic influence in relation to accidents, we find that the stick is entirely too short. Many drivers, definitely impaired in their driving abilities after indulgence in alcohol, escape entering our statistics on alcohol and accidents. The reason for this is that those persons engaged in law enforcement have conveniently failed to recognize physical symptoms associated with the influence of alcohol, and have failed to recognize that a person under the influence of alcohol, even though capable of walking a chalk line, is just as serious a menace on the highway as the person who is out and out drunk. In these cases a charge of "reckless driving" or "driving too fast for conditions" is applied. Thus the facts and figures on the relationship of alcohol to accidents are too conservative.

Under Chapter 95½, Section 144 of the Illinois Statutes, 1955, we find the following:

(a) It is unlawful and punishable as provided in subdivision (b) of this section for any person who is an habitual user of narcotic drugs or any person who is under the influence of intoxicating liquor or narcotic drugs to drive any vehicle within this State.

(b) Every person who is convicted of a violation of this section shall be punished by imprisonment for not less than 2 days nor more than 1 year, or by fine of not less than $100 nor more than $1000 or by both such fine and imprisonment. On a second or subsequent conviction he shall be punished by imprisonment for not less than 90 days nor more than 1 year, and, in the discretion of the court, a fine or not more than $1000.

Apparently our General Assembly in the passage of this Act was fully aware of the fact that alcohol and narcotics in general (except cocaine) have something in common. Both are depressants. Therefore, in coupling alcohol to narcotics under this section, our legislators have

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*Presented at a lecture sponsored by The Chicago Medical School.

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in effect stated that a narcotic or alcohol is a substance which induces sleep, relieves pain, causes a stupor, and gives rise to addiction. Thus the narcosis produced by the depressant action of alcohol or narcotics of necessity impairs one's ability as a driver on the road. This impairment is manifest by way of a warped judgment and a lack of muscular coordination. So, instead of finding a fully capacitated and alert driver at the wheel, we find nothing more than a torso. Certainly more is required for the sane operation of a motor vehicle.

Unfortunately, the statute fails to set forth any suitable standards by which influence can be measured. Therefore, we are relegated to an opinion of a police officer that a driver was under the influence of intoxicating liquor because of a traffic error and abnormal behavior following, or the inability of the driver to adequately express himself. Conceding the traffic error, should abnormal behavior or the inability of adequate expression be the chief basis for an opinion regarding alcoholic influence?

Definitely not, when there is a possibility that one of sixty pathological conditions or even fright through sudden police contact can bring about the characteristics forming the ground for the opinion of alcoholic influence.

Obviously convictions based on speculative and conjectural testimony are grossly unfair. So, in order to vigorously prosecute the guilty, and in order to protect the innocent, we must resort to the employment of chemical tests to aid in diagnosing influence and/or intoxication.

The American Medical Association through one of its committees (JAMA, May 27, 1939, page 2165), has taken the following position:

> The concentration of alcohol in the blood is one of the best criteria of intoxication because blood alcohol concentrations closely parallel detrimental effects noted in carefully conducted experimental tests. Although this relationship is not mathematicalily exact because of slight variations resulting from inherent differences in human beings, it is sufficiently accurate for practical purposes. The relationships between concentrations of alcohol in blood, urine, saliva and breath have been shown to be sufficiently definite so that chemical tests of any of these body materials can furnish a reliable measure of the degree of alcoholic influence.
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> For medicolegal purposes, the committee recommends the following interpretation of chemical tests for alcohol:
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> 1. Although there is no minimal figure which can be set at which there will be absolutely no effect from alcohol, the committee recommends that persons with a concentration of alcohol of less than 0.05 per cent by weight in blood or its equivalent in urine, saliva or breath should not be prosecuted for driving while under the influence of alcoholic liquor.
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> 2. All persons show a definite loss of that clearness of intellect and control of themselves which they would ordinarily possess when the concentrations are above 0.15 per cent in the blood or its equivalent in other body fluids or breath and should therefore be considered as under the influence.
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> 3. When the alcohol concentrations are between 0.05 and 0.15 per cent in the blood, a great many of the persons will be under the influence of alcohol, but the committee recommends prosecution only when the circumstances and results of physical examination give definite confirmation of such influence.

Statutes in some seventeen or eighteen states have followed verbatim the limits and degrees of intoxication as outlined by the American Medical Association. However, it is significant to note that the State of Illinois has failed to adopt similar legislation. Therefore, the question arises, are chemical tests for intoxication admissible in evidence in the absence of specific statute?

The answer to the question in the affirmative lies in a decision of the Appellate Court of the State of Illinois, People v. Bobczyk, 343 Ill. App. 504. The Court in upholding the judgment of the trial court held that the defendant was under the influence of intoxicating liquor to the extent that his driving ability was impaired, that the Drunkometer Breath Test is admissible, and that the State is not compelled to rely solely on the opinions of non-expert witnesses to prove the offense charged.

It was my pleasure and my duty in 1949 to participate in the test case in conjunction with Dr. Harger, Professor of Biochemistry and Toxicology at the Indiana University School of Medicine, Indianapolis, Indiana, Dr. Muehlberger, Chief Toxicologist, Michigan State Board of Health, East Lansing, Michigan, and Mr. Robert Donigan, Chief Counsel, Northwestern University Traffic Institute.

Several questions and answers on direct and cross examination may be of interest.

Q. Knowing the blood alcohol concentration, can you state the quantity of alcohol accumulated in the body in terms, say, of whisky?

A. Yes. A blood alcohol concentration of .15 per cent in a person weighing 150 pounds indicates the accumulation in his body of the alcohol contained in 6 ounces of whisky; a blood alcohol concentration of .20 percent would indicate the accumulation in the body of the alcohol contained in 8 ounces of whisky.

Q. In the present case, did you conduct a Drunkometer test for intoxication on defendant's breath?

A. Yes, I did.

Q. When and where was the test conducted?

A. On October 23rd, 1949, at the Chicago Police Scientific Crime Detection Laboratory, 1121 S. State Street, Chicago, Illinois.

Q. What did the test indicate?

A. It indicated that defendant had been drinking.

Q. What concentration of blood alcohol did you find?

A. .30 percent.

Q. The percentage that you indicated represents the accumulation in the body of how much alcohol?

A. The percentage indicated represents the accumulation in the body of the alcohol contained in 10 ounces of whisky, or in 10 bottles of beer.

Q. Would this person have to drink more than that amount of whisky or beer to show a blood alcohol percent of .30?

A. Yes, because he would have burned some alcohol in the accumulation period.

Q. From the results of the test have you formed an opinion as to whether or not this defendant was under the influence of alcohol at the time of his arrest?

A. Yes, I have.

Q. What is that opinion?

A. That the defendant was under the influence of alcohol to the extent that he had lost some of that clearness of intellect and control of himself which normally he would possess.

Cross Examination

Q. Suppose a man had some drinks and a period of time elapses, what happens to the alcohol?

A. The alcohol is oxidized.

Q. Can you tell from your own knowledge using the Drunkometer how long it would take a man to get rid of an alcoholic percentage of .30?

A. Using the Drunkometer I do not know how long it would take for a man to get rid of .30 percent of alcohol. However, I do know from experience and experimentation that one loses about two-thirds of an ounce of whisky per hour through oxidation.

Q. Can you tell from the Drunkometer how long it has been since the individual had anything to drink?

A. No.

Q. How much alcohol is reacted upon in this test?

A. .169 milligrams.

Q. How much air produces .169 milligrams of alcohol?

A. The volume of air necessary to produce .169 milligrams of alcohol will vary with every concentration of blood alcohol.

Q. Your findings could not be right or there must have been something wrong with your apparatus if the man said that he had two beers?

A. They all say that they had two beers.

Q. Just answer my question. Would your findings show .30 percent if he had only two beers?

A. No. That is impossible.

Q. Would it show .30 per cent if he had three ounces of whisky?

A. No.

Q. In other words, you are quite definite that he must have had at least 10 bottles of beer or 10 ounces of whisky?

A. That is right.

Q. Now with reference to this test that you gave him, Mr. Dragel, as a matter of fact he refused to take the test?

A. He at no time refused to take the test.

Q. As a matter of fact, you told him that he must take the test?

A. On the contrary, I advised him of his constitutional rights.

Defense Counsel: Maybe the test is fallible, maybe the defendant had only two beers.

The Court: I have heard hundreds of people testify and that seems to be the stock answer, two glasses of beer. I am surprised that all whisky companies have not gone bankrupt because that is all people drink, beer.

Other testimony in the case outlined in considerable detail the operation of the Harger Drunkometer and the calculation of the percent of alcohol in the blood through analysis of breath. It follows in part:

A sample of breath from a balloon is attached to an inlet tube and allowed to bubble through a reaction tube. The reaction tube contains one cubic centimeter of one-twentieth normal potassium permanganate and approximately ten cubic centimeters of fifty-six percent sulfuric acid. The alcohol in the breath is caught by the acid and causes a reduction of the purple permanganate solution. The chemical change is a decisive one marked by the disappearance of the purple color and the formation of a straw color which is compared with a standard. At this end-point one cubic centimeter of permanganate solution has reacted upon .169 milligrams of alcohol, and the volume of breath needed to furnish .169 milligrams of alcohol is measured in a sealed container called a gasometer by water displacement.

Since 2100 cc. of deep lung breath or 3200 cc. of expired breath contain the same weight of alcohol as 1 cc. of blood, the concentration of alcohol in the blood is calculated by simple proportion.

$$.169 \div 190 \div X \div 3200$$

X equals 541

290

X equals 1.86 milligrams per cc or .19 per cent.

(290 is the assumed volume of each breath)

The second part of the test also measures the volume of breath used. The breath leaving the reaction tube i spassed through a drying tube and then into an ascarite tube, its purpose being to absorb the carbon dioxide in the breath. The increase in the weight of the ascarite tube is the amount of carbon dioxide absorbed. Since 3200 cc. of expired breath contains 190 mgs. of carbon dioxide, the amount of breath used can be again determined by simple proportion.

$$.169 \div 16.9 \div X \div 190$$

X equals 32.1

16.9

X equals 1-.89 milligrams per cc. or .19 percent.

(16.9 is the assumed weight in mgs. of the volume of CO_2 absorbed.)

At the laboratory I am requested by various agencies throughout the state to determine the amount of alcohol in a given sample of blood. For this purpose I usually employ Dr. Harger's micro-method for the detection and determination of alcohol in the blood, or the method outlined by Levine and Bodansky in a supplement of the American Journal of Clinical Pathology. The chemistry of both methods are similar in that both employ acid potassium dichromate as the oxidizing agent, the excess of dichromate being titrated with a ferrous sulfate, methyl orange solution.

In the trial of cases involving charges of driving while under the influence of alcohol many objections have been raised to the admission of chemical evidence of intoxication. Foremost is the constitutional privilege against self-incrimination. While superior court decisions in many states differ, the generally accepted view is that the taking of breath, urine, or blood for the purpose of determining the percentage of blood alcohol of a defendant is not a denial of the constitutional privilege.

In State v. Morkrid, 286 N.W. (2) 412. Iowa, 1939, the Court held that results of chemical tests of specimens voluntarily given by a defendant charged with driving while intoxicated with his expectation that the results would show he was not intoxicated, were admissible to show his intoxication, and there was no violation of the defendant's privilege against being compelled to be a witness against himself.

Consent to taking a chemical test for intoxication waived the constitutional privilege. But, does an intoxicated person possess the mental capacity to give consent to a waiver of a constitutional privilege?

Yes, according to a decision of the Court in Halloway v. State, 175 S.W. (2) 258, Texas, 1943. In this case it was held that results of a chemical test of specimen voluntarily given by a defendant charged with driving while intoxicated after a police officer told him he did not have to give specimen, that it would be analyzed for alcoholic content, and that results thereof might be used against defendant, and expert testimony of chemists as to effect of various percentages of alcohol upon an individual in whose urine it is found, were admissible. The fact that defendant may be intoxicated does not necessarily render him incompetent to give consent to taking a specimen for chemical analysis and thereby waive any constitutional rights which he may have.

While the decisions in these two cases follow the general rule that the taking of a specimen for the purpose of determining the percentage of blood alcohol of a defendant in a drunken driving case is not a denial of the constitutional privilege, we find a better judicial expression of the applicable law on the subject in the following:

In Ridgell v. United States, 54 A. (2) 679, Washington, D.C., 1947, the court held that the results of a chemical test of specimen (urine) voluntarily given by defendant charged with negligent homicide after a police officer warned him that results of such test could be used for or against him were admissible and there was no violation of any rights guaranteed by the Constitution. The whole history of the privilege against self-incrimination shows it was designed solely to protect against compulsory testimony.

In the cited cases the defendant voluntarily gave up the required specimen. However, assuming that a defendant was unconscious and bleeding after a serious accident, would the taking of his blood while he was unconscious on the emergency table for the purpose of determining blood alcohol be a denial of any other constitutional privileges?

In State v. Weltha, 292 N.W. 148, Iowa, 1940, the Court held that results of a chemical test of a specimen (blood) taken from the defendant while he was unconscious and before his arrest for manslaughter by a motor vehicle, which was made later upon basis of such test

results, were not admissible because taking of specimen was an unlawful search and seizure. The evidence in this case also was insufficient to establish the identity and condition of the specimen.

In another case, Rochin v. California, 342 U.S. 165, 1952, while the subject matter did not relate to alcohol, the decision of the court had a far-reaching effect on physical evidence in general.

The court held that evidence obtained from the body of the defendant (contents of stomach containing narcotics recovered by forcible use of stomach pump) by such force and brutality that even hardened sensibilities are offended is inadmissible because it constitutes a violation of the due process clause of the fourteenth amendment to the United States Constitution. In deciding this case, we do not heedlessly bring into question decisions in many states dealing with essentially different, even if related, problems. We, therefore, put to one side cases which have arisen in the state courts through use of modern methods and devices for discovering wrongdoers and bringing them to justice. It does not fairly represent these decisions to suggest that they legalize force so brutal and so offensive to human dignity in securing evidence from a suspect as is revealed by this record.

In chemical test cases, too, we are often confronted with a rule of evidence known as a privileged communication, or the physician patient privilege. The privilege was unknown at common law but is now embodied in the statutes of more than half of the states. The purpose of the particular statute is to protect the confidential relationship existing between doctor and patient. The privilege is invoked as a defense or as a legal objection to the disclosure of evidence of intoxication which has come to the knowledge of the physician. In cases where the privilege has been upheld, the doctor was called to treat the patient for injuries. Thus the relationship was established.

However, in Hanlon v. Woodhouse, 113 Colo. 504, (1945), where a doctor was requested by police officers to draw a blood specimen from an unconscious person for the purpose of testing for alcoholic content, and even though the doctor treated the person for injuries, there was no violation of the rule concerning privileged communications because information thus obtained was not necessary to enable the physician to prescribe or act for the patient.

SCHOOL NOTES AND NEWS

FACULTY APPOINTMENTS

Dr. John J. Sheinin has announced the following appointments to the faculty of The Chicago Medical School:

Department of Anatomy

Dr. John F. Pauly as Instructor in Gross Anatomy.

Dr. James E. P. Toman as Assistant professor of Anatomy and Assistant Professor of Physiology.

Department of Pathology

Dr. Stephen R. Greenberg as Instructor.

Dr. Jonathan B. Horrell as Clinical Instructor.

Dr. Marija Mackeviciute as Assistant.

Dr. Paul B. Szanto as Associate Professor.

Department of Medicine

Dr. James B. Berardi as Clinical Instructor.

Dr. Gerald H. Becker as Clinical Instructor.

Dr. Lester Cohn as Clinical Assistant.

Dr. Morton Einhorn as Clinical Assistant.

Dr. Edwin Feldman as Clinical Assistant.

Dr. Irving Forman as Clinical Assistant.

Dr. Arnaldo Libretti as Research Fellow in Allergy.

Dr. Stanley Leithold as Clinical Assistant.

Dr. Fred Levit as Clinical Assistant.

Dr. William C. Maslow as Clinical Instructor.

Dr. Jerome J. Podgers as Clinical Assistant.

Dr. Lester Rice as Clinical Associate.

Dr. Stella B. Riskus as Clinical Assistant.

Dr. Lee H. Schlesinger as Clinical Associate Professor.

Dr. Joseph L. Sokolov as Clinical Assistant.

Dr. Alexander B. White as Assistant.

Department of Neurology and Psychiatry

Dr. Joseph W. Friedlander as Associate in Psychiatry.

Dr. Irving C. Sherman as Clinical Professor of Neurology and Chief of Division of Neurology.

Dr. Earl N. Solon as Clinical Assistant.

Department of Pediatrics

Dr. Minnie Frank as Clinical Assistant.

Dr. Seymour Metrick as Clinical Assistant.

Department of Radiology

Dr. Bertram Levin as Clinical Assistant Professor.

Department of Surgery

Dr. Peter Beaconsfield as Assistant Professor.

Dr. Julius Brant as Clinical Assistant.

Dr. Helen Button as Clinical Instructor.

Dr. Jean F. Conte as Clinical Instructor.

Dr. Harry H. LeVeen as Associate Professor.

Dr. Joseph M. Levenson as Clinical Instructor.

Dr. Manfred S. Prenzlau as Clinical Associate (Anesthesiology).

Dr. Earl B. Sanborn, Jr., as Associate.

Department of Orthopedic Surgery

Dr. Philip Falk as Instructor.

Dr. Sydney H. Morgenstern as Clinical Assistant.

Dr. Maurice S. Stamler as Clinical Associate.

Department of Neurosurgery

Dr. Paul R. Rosenbluth as Clinical Instructor.

Department of Otolaryngology

Dr. Douglas W. Mazique as Clinical Instructor.

Department of Obstetrics and Gynecology

Dr. David D. Turow as Clinical Assistant.

Dr. Seymour D. Wishnick as Clinical Assistant.

PROMOTIONS

Dr. Sheinin has also announced the following faculty promotions:

Department of Anatomy

Dr. John J. Chiakulas to Associate in Gross Anatomy.

Department of Dermatology and Syphilology

Dr. Aaron L. Goldberg as Associate.

Department of Medicine

Dr. Meyer J. Barrash to Associate.

Dr. Amos Brown to Clinical Instructor.

Dr. Albert Cohan to Clinical Instructor.

Dr. Louis Richmond to Associate in Medicine and Acting Director of Mt. Sinai Dispensary.

Dr. Louis H. Turek to Clinical Instructor.

Dr. Philip Warsaw to Clinical Instructor.

Dr. Everett G. Weir to Clinical Associate.

Department of Neurology and Psychiatry

Dr. Charles Myran as Associate.

Department of Pediatrics

Dr. Lester Wishingrad to Instructor.

Department of Surgery

Dr. Robert D. Crane to Clinical Associate.

Dr. Ellsworth E. Hasbrouck to Clinical Associate.

Dr. Alfred W. Kneucker to Assistant Professor.

Dr. Herbert D. Trace to Clinical Instructor.

Department of Urology

Dr. Howard H. Bass to Clinical Associate.

Department of Obstetrics and Gynecology

Dr. Howard I. Ganser to Clinical Instructor.

Dr. Maurice J. Golden to Clinical Instructor.

Dr. Sidney R. Lash to Clinical Instructor.

Dr. Lawrence LeVine as Clinical Instructor.

Dr. Joseph S. Poticha to Clinical Associate.

Dr. Leonard P. Rapoport to Clinical Instructor.

Dr. Jerome B. Reich to Clinical Instructor.

Dr. Harold E. Silverman to Clinical Associate.

Dr. Maxwell Wacker to Clinical Instructor.

Dr. Victor M. Williger to Clinical Instructor.

Dr. Howard L. Woolf to Clinical Instructor.

Dr. Jack Zackler to Clinical Instructor.

REAPPOINTMENTS

Dr. John J. Sheinin has announced the following reappointment to the faculty of The Chicago Medical School:

Department of Surgery

Dr. Eugene A. Noskin as Assistant.

CLASS NEWS

Class of 1956

Congratulations to Donald Winikoff, of the Bronx, N. Y., on the occasion of his marriage to the former Evelyn R. Weiss, of Brooklyn, N. Y., on June 26, 1955.

Best wishes to Stanley Renner of Far Rockaway, N. Y., on the occasion of his marriage to the former Maureen Marin, of Far Rockaway, on September 4, 1955.

Congratulations to Arnold Sadwin, of Northfield, R. I., on the occasion of his marriage to the former Susie Mae Matney, of Matneytown, Oceana, W. Va., on November 24, 1955.

Best wishes to Richard Helfman, of New York, N. Y., on the occasion of his engagement to Susan Beth Sherman, of Chicago, Ill., on November 25, 1955.

Congratulations to Paul Kiell, of Newark, N. J., on the occasion of his engagement to Benita Seifer, of Chicago, Ill., on December 9, 1955.

Congratulations to Roslyn and Alan Bloom on the birth of their son, Jordan Mark, on December 12, 1955.

Best wishes to Joseph T. Harris, of Forest Hills, N. Y., on the occasion of his marriage to the former Betty Ruth Marks, of Chicago, Ill., on December 11, 1955.

Class of 1957

Congratulations to Sherwin and Charlene Kornblum on the birth of their daughter, Candyce Jill, on November 29, 1955.

Congratulations to Edward B. Magid, of Chicago, Illinois, and Miss Naoma Ruth Deutsch, also of Chicago, on the recent announcement of their engagement.

Congratulations to Gwen and Gene Schwartzman on the birth of their daughter, Shari Lee, on January 16, 1956.

Congratulations to Jerry and Ruth Siegel on the birth of their son, Andrew L. Siegel, on September 13, 1955.

Class of 1958

Congratulations to Francis Stein on the occasion of his engagement to Miss Marilyn Rosen, of Newark, N. J.

Class of 1959

Congratulations to Joel Kaplan on the occasion of his marriage to the former Miss Roberta Rifkin, of Saginaw, Mich.

Congratulations to Irwin Scher on the occasion of his marriage to the former Myra Mendelson, of New York City.

Congratulations to Harold Weiss on the occasion of his engagement to Miss Barbara Geller, of Rockford, Ill.

Congratulations to William Werner on the occasion of his engagement to Miss Rosemarie Coviello, of Far Rockaway, N. Y.

Congratulations to Henry Nachamie upon his engagement to Miss Sandra Epstein, of Peekskill, N. Y.

ABSTRACTS SECTION

LUISADA, ALDO A. (Assoc. Prof. of Medicine; Director, Division of Cardiology), and DIAMOND, IRA (Research Fellow). Action of Cardiac Glycosides on Diastolic and Resting Length of Cardiac Strips. Amer. J. of Physiology, *181*:347-351, May, 1955.

The effects of thirteen cardiac glycosides on resting and diastolic length were tested on preparations of ventricular strips contracting isotonically. Striking differences were noted. Ouabain and acetyl-strophanthidin were the most powerful and caused shortening of the strips. Gitalin was slightly less effective in high dilutions but had a remarkable range of action. Thevetin, scillaren B, digitoxin, digoxin, acetyl-scillirosidin, and acetyl-digoxin were in a third, somewhat less effective group. Lanatoside C and Acetyl-digitoxin were in a different group because they caused shortening only at high concentrations. Several drugs caused relaxation in low concentrations, below those existing in the blood of fully digitalized patients. On the other hand, acetyl-digitoxin caused relaxation in clinical concentrations while contraction was caused by concentrations which would be equivalent to clinically toxic doses. The electrical manifestations of the ventricular strip were studied.

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ZILLA, A. (Research Assistant) and LUISADA, A. A. (Assoc. Prof. of Medicine, Director, Division of Cardiology). Effects of Acetyl-Digitoxin alpha in Ambulatory Patients with Congestive Failure. Experimental Medicine and Surgery, *13*:2, 3-8, June, 1955.

The action of acetyl-digitoxin alpha, a derivative of lanatoside A, was studied in 19 ambulatory patients in failure. Slow digitalization (2 to 6 days) was obtained with doses of 2.4-3.6 mg. The maintenance dose varied between 0.1 and 0.4 mg. Clinical, electrocardiographic, roentgenological, and functional observations were made.

Toxic phenomena were observed in 3 cases. Minor disturbances occurred in another two.

A comparison between acetyl-digitoxin and digitoxin revealed a basically similar, occasionally slightly superior, therapeutic effect of acetyl-digitoxin.

While the toxic effects were found similar to those which may be caused by digitoxin, they were less severe and their disappearance was obtained within a shorter time on account of more rapid elimination of the drug. Therefore, Acetyl-digitoxin alpha was found easier to administer, especially in ambulatory cases.

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PRESMAN, DAVID (Clin. Assoc. Prof. of Urology). Clinical Experiences with Retropublic Prostatectomy. J. of the International College of Surgeons. 23:2, 232-240. February, 1955.

Clinical experiences with retropubic prostatectomy based upon a large series of cases justifies the general conclusion that this is a sound surgical procedure for enucleation of the benign hypertrophied prostate.

The basic advantage of this operation over the other approaches to the prostate is the improved exposure of the operative field, which permits a more thorough removal of all obstructive tissue and improved hemostasis under direct vision.

Postoperative complications, although less frequent, have not been completely eliminated and will require further experience and study on the part of urologists if the postoperative results are to be improved.

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MANN, LAWRENCE S. (Assoc. in Surgery), KALLEN, IRWIN A. (Assistant in Surgery), TOMUSK, AUGUST, FRIEDMAN, FRANKLIN P. Rupture of the Stomach in the Newborn Infant with Survival. Surgery, 37:8, 969-972. June, 1955.

A case of rupture of the stomach in the newborn infant, secondary to a congenital defect in the musculature, occurring 45 hours after birth is presented. The authors believe this to be the fifth case reported in the literature which has survived. Prompt diagnosis and early operation to correct this defect are believed to be the main factors for survival. A distended abdomen, absent bowel sounds and free mobility of the large bowel on rectal examination are presumptive findings of a ruptured viscus which can be corroborated by x-ray examination in the upright position. The presence of a pneumoperitoneum with air in the small and large bowels and the absence of an air bubble in the stomach should be diagnostic of a ruptured stomach.

* * * * *

LUISADA, ALDO A. (Assoc. Prof. of Medicine; Director, Division of Cardiology) and CARDI, LUIGI (Research Assistant). Further Studies with Antifoaming Agents in Experimental Pulmonary Edema, Circulation Research, *3*:5;510-513.

The effect of several antifoaming agents by inhalation in preventing acute pulmonary edema caused by intravenous epinephrine was tested in 153 rabbits.

Three agents were definitely beneficial: 10 per cent silicone in water, freon, and ethyl alcohol. When inhaled alcohol has mild systemic effects, silicone and freon (in short periods of admin istration) have none. Therefore, the effectiveness of antifoaming therapy in general was confirmed

Mixtures of antifoaming agents, tested in search of a possible synergistic effect, did not prove superior to the single agents. Even 10% ether in alcohol, in spite of some sedative effect, did not prove to be more effective than alcohol.

As epinephrine pulmonary edema is different from several types of clinical pulmonary edema, and freon should be excluded because it might have toxic effects if administered for several hours, a clinical trial with silicon (aerosol) and ether-alcohol (aerosol), seems indicated.

THE CHICAGO MEDICAL SCHOOL

QUARTERLY

710 SOUTH WOLCOTT AVENUE

Chicago 12, Illinois

The Chicago Medical School *QUARTERLY* is published four times yearly by the Chicago Medical School for the dissemination of current medical news and for the advancement of medical science with a student staff under the supervision of a faculty editorial board.

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